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Anaesthesia

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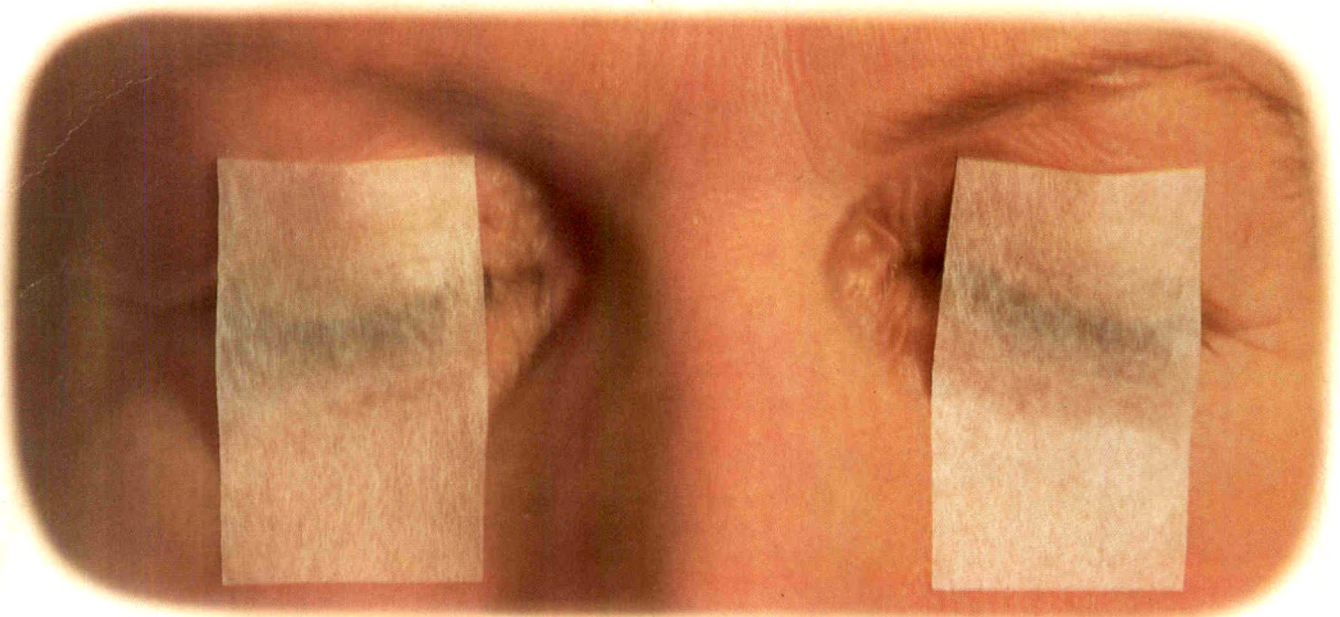


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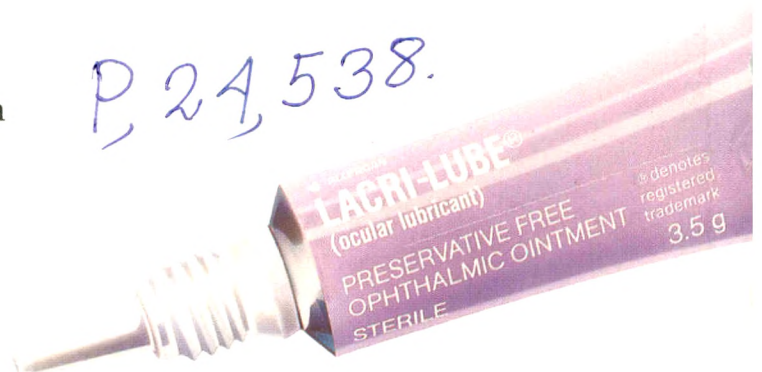
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Curr Res 1977; **56** (1): 35-37. 2. Snow, J.C. et al., *Anesthesia and Analgesia* ...
Curr Res 1975; **54** (4): 465-467.

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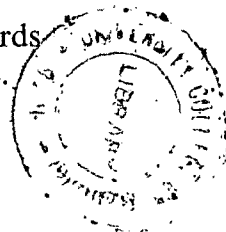
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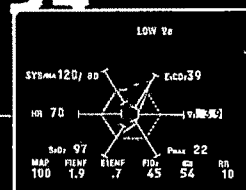
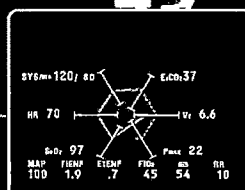
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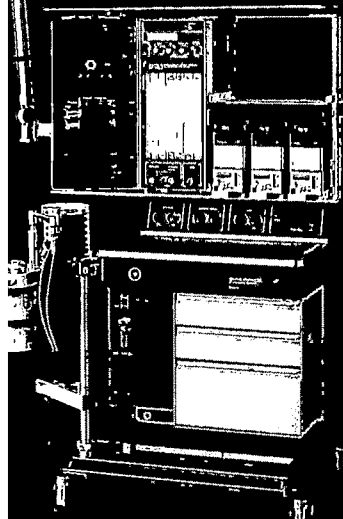
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The Royal College of Anaesthetists

The Association of Anaesthetists of Great Britain and Ireland congratulates and salutes the Royal College of Anaesthetists on obtaining an independent Charter and the acknowledgment from Her Majesty, Queen Elizabeth II.

The event will prove to be an historic landmark for our young specialty for it is exactly 150 years since Crawford Long administered the first anaesthetic in Atlanta, Georgia, USA. In those 150 years anaesthesia has been the single most important element in facilitating advances in patient treatment, safety and comfort. Without anaesthesia surgery would be at a standstill, without anaesthesia obstetrics would be horribly primitive, without the skills of anaesthetists patients would not be relieved of pain nor be resuscitated from major trauma and cardiac arrest. All of the critically ill would die. It can be said truly that we have won our spurs and deserve the accolade granted by the Privy Council in our independent Charter and the honour bestowed upon us by Her Majesty in conferring the Royal title.

The Association takes particular pleasure and pride in the achievement for it was that body's foremost aim when it was created 60 years ago under the leadership of its first President, the redoubtable Henry Featherstone. Throughout its existence, the Association has remorselessly strived to ensure that anaesthesia is recognised as a branch of medicine at least equal in status to all other specialities. The initial step came 44 years ago with the creation of the Faculty of Anaesthetists, brought about by a combination of pressure and negotiation by the Association, the support of some far sighted and wise surgeons and by the example set by anaesthetists in the previous decade during the years of the second World War.

Some 25 years ago the concept of an independent College of Anaesthetists was first mooted and became a hotly debated topic in the 1970s. There were some who thought it preferable not to cut the umbilical cord attaching us to the prestigious and powerful Royal College of Surgeons of England. It has to be said that many of those who doubted were in fact members of the Board of Faculty at that time — perhaps because from the inside they, above all, could see the apparent benefits of

remaining within a recognised bastion of the medical hierarchy. Fortunately the leadership and an increasing majority of the members of the Association, supported wholeheartedly by the Junior Anaesthetists Group, tenaciously stuck to their goal of true independence for a speciality of which they were inordinately proud. Over a quarter of a century the tide of opinion turned to unanimous support and it has to be said that none could have done more to work for an independent College than Professors Michael Rosen and Michael Vickers with the support of visionary figures such as Drs Philip Helliwell, Harry Churchill Davidson, Derek Wylie and John Zorab. They deserve our thanks and admiration as do our colleagues on the Council of the Royal College of Surgeons led by Sir Geoffrey Slaney, Sir Ian Todd and Sir Terence English. They have been totally understanding of our aspirations and have spared no effort to support us in our quest. They epitomise the attitude of the modern surgeon who is wise enough to realise the potential and the power of the equal partnership that has arisen between our two specialties. It is a matter of great satisfaction that our new status has been applauded by all of our sister colleges, by the British Medical Association and by the Department of Health.

Thus, 1992 is a very proud year for anaesthesia, heralding both the Diamond Jubilee of the Association and the inception of the Royal College of Anaesthetists. March is a fortuitous month for our specialty. It is the month of the creation of our Faculty. Forty-four years later in the same month almost to the day our Royal College was born. The sign of the Zodiac for this anniversary date is Pisces. The fish is a symbol of good fortune. The Association wishes the Royal College the good fortune it has earned and deserves. We offer them, as ever, total support in their quest to further the standard of care of those who really matter — our patients.

*President
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Editorial notices

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Editorial

Persuasion in clinical practice: rational decision or intuitive belief?

Medicine makes progress in two general ways: from the information that accrues from the collection of data, and from the interpretation of information by the construction of theories. The theory may precede the information; some theories may remain unproven or become redundant; but there is danger in information without theory. We like to think that our actions in clinical practice are based on sound scientific observations and reasoning, but much is done out of habit, or for empirical reasons, or because it was accepted as correct many years ago and has never been questioned. Specialists in all branches of medicine can give examples.

The controlled clinical trial is the recognised structure for framing questions intended for the rational improvement of clinical practice, although doctors have a poor record of taking notice even of well constructed trials [1] while at the same time tending to take undue notice of what is effectively hearsay evidence [2]. In anaesthesia, progress has followed a better understanding of physiology and pharmacology, with the introduction of new drugs, of new techniques, and of new equipment. Clinical trials, if suitably framed, are well suited to investigations of new drugs and can work well with new techniques, although there is then the almost inevitable problem of bias caused by difficulties making observations and patients truly blind to the choice of treatments.

The framing of a clinical trial—in other words, the collection of objective data—can become much more difficult when the question is whether outcome is improved by new equipment. New equipment demands investment by the companies who make it and its potential benefits are usually extolled by the enthusiasts who are its first users. Patients in clinical trials do better than similar patients not in trials [3], so it is not surprising that new equipment, particularly if it requires an operator to be with the patient, is perceived as improving outcome. The more expensive the equipment, and the more ill the patient, the more difficult a proper evaluation becomes. The more exposure an innovation is given, the more difficult it becomes to question its utility [4, 5]: 'The emotions caused by apparently early benefits in inadequately designed studies may otherwise overpower reason.' [5].

If the new equipment is not directly curative but is instead a source of new information, then it becomes even more difficult to resist early impressions that collecting this information is an improvement in clinical management. In our specialty, the microprocessor and allied technology allow us to measure more and more variables more and more frequently, particularly in the intensive care unit. The new technology certainly gives us more to look at and may give us more to do; but does this necessarily confer benefit on the patient—more benefit, that is, than conferred by having a concerned clinician at the patient's side?

One such innovation is the continuous measurement of the oxygen saturation of mixed venous blood ($S\bar{V}O_2$). An article, circulated by a manufacturer of the equipment but printed under the auspices of the Northeastern Intensive Care Society (based in Newcastle, England) in November 1990, is an example of what I believe to be false logic in promoting widespread use of new techniques.

After a few pages of useful discussion of physiology and of the practical application of the pulmonary artery catheter, the writer, a British intensivist now resident in the United States, addresses the question, 'What is this all for?'

'It is more difficult to demonstrate that having this information available produces an improved outcome. Despite the work of Rao, and Schultz at Valhalla few other clear cut studies suggesting an influence on outcome can be measured with [the] current level of understanding and available therapies.'

This is as clear a statement of current knowledge as one could wish. There are two possible routes for the progression of the argument: that more studies need to be done; or that, at the least, it is a technique that is useful in teaching. Instead, the acknowledged lack of evidence is ignored and critics of the technique are attacked.

'There is a small but stridently vocal group of critics which routinely decries "The Cult of the Swan-Ganz Catheter". These shrill assertions, generally supported by even less data than the studies they seek to attack, must in my view assume the burden of proof. In other words it must be shown that the avoidance of any morbidity associated with the use of right heart catheters produces a net improvement in patient outcome!'

Note the pejorative terms 'stridently vocal' and 'shrill assertions'; and the exclamation mark carries the implication that it is absurd to suppose that the risks can possibly outweigh the benefits. (The parenthesized quote was the title of an article.)

Much more importantly, the burden of proof has been turned around. It is emphatically *not* the task of doubters to provide counterproof of the value of a new therapy or technique; it is the task of the believers to provide solid evidence of efficacy. The writer continues: 'The wise clinician would rarely opt for a situation which gave him *less* information not more.' [author's italics] This is not so. We no longer, for instance, request X rays before routine abdominal surgery. Information must not be sought for its own sake; the wise clinician seeks information only when it is useful [6].

Having attempted to justify the use of the pulmonary artery catheter despite considerable evidence that it confers no real benefits [7], the author recommends the routine use of continuous monitoring of $S\bar{V}O_2$ in all critically ill patients. The recommendation is based on a

single study in his own hospital, which included only patients who had undergone cardiac surgery, and for which the only reference cited is an abstract [8].

The same has happened with a method using bio-electrical impedance to estimate total body water. The company circulated an abstract of an accepted but at the time unpublished paper in which this method was compared with isotopic dilution. No data are provided in the abstract; the only figures given are the statistics resulting from a presumed least squares regression, a common but misleading practice [9]. The accompanying letter says of the equipment that 'its applications appear almost limitless . . . it has become apparent that one of those applications is in critical care situations where the Analyser's ability to monitor total body water non-invasively yet simply and speedily has obvious attractions (see enclosed abstract . . .)'. The only situation tested was paediatric cardiac intensive care; the sample was of 35 unspecified children; and the study was not of the utility of the measurement but of how the measurement compared with a previous method.

It will be good if the companies' publicity encourages research on how these measurements affect outcome, and it may turn out that one or both are of genuine benefit to critically ill patients; but medical resources for clinical treatments must not be spent on unproven techniques or on assumptions that measurement is outcome. The mistaking of physiology for outcome is responsible for the importance attached to the haemodynamic response to intubation [10], and it has caused much confusion in the treatment of myocardial ischaemia [11]. There, the picture has been further clouded by sometimes dubious financial interests [12], interests that did irreparable harm in the early stages of the artificial heart programme [13].

However good the promise, clinical practice must not be based on single abstracts or on the good intentions of well meaning enthusiasts, who are not unnaturally protective and supportive of their own ideas and efforts. In an early abstract about oesophageal motility [14], alveolar concentration of halothane and a clinical score of anaesthetic depth were reported as correlating 'very highly' with measures of motility. In a full paper, by which time this phrasing could have been used for 2 years to promote the value of the technique in clinical anaesthesia, the investigators wrote rather circumspectly that motility 'may have some potential value as a means of supplementing existing clinical signs' [15] but the results of research since then have been disappointing.

In the field of physical chemistry Fleischmann and Pons believed they had discovered a cheap and endless source of energy from nuclear fusion in a test tube. No one can deny their enthusiasm or strength of belief, and the world would have been transformed (and many fortunes made) had they been right. But they were not; their experiments were flawed and their results incorrect [16].

Putative advances in anaesthesia are unlikely to receive the attention that cold fusion received, but the lessons in that episode for the world at large are there for us as individual anaesthetists. New techniques, whether in treatment or monitoring, must not become common currency just because everything must be done for the dying patient, or because enthusiasts are convinced that only good can come of them, or because

of the novelty of measuring something not previously measurable. When treating desperately ill patients the easy course of action is to do something [17]; it takes courage to admit that there is nothing that can or should be done [18]. It is not easy to make sensible decisions about complex technical issues [19], but we must keep a firm grip on what facts we have and not let our impatience with the difficulties of the real world divert us from thinking logically about them.

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Recovery characteristics using isoflurane or propofol for maintenance of anaesthesia: a double-blind controlled trial

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Summary

We studied 114 female patients (ASA 1 or 2) who were within 20% of ideal body weight and who were scheduled to undergo gynaecological laparoscopy which required supplementation with an opioid (groups IA and PA), or dental procedures which did not require opioid supplementation (groups IO and PO). A computerised package of psychomotor tests was performed before surgery. Anaesthesia was induced with propofol 2.5 mg.kg^{-1} and all patients received atracurium 0.3 mg.kg^{-1} and 67% nitrous oxide in oxygen. Patients in group IA received isoflurane 1% (inspired), and alfentanil $10 \text{ }\mu\text{g.kg}^{-1}$ as a bolus and $10 \text{ }\mu\text{g.kg}^{-1}.\text{h}^{-1}$ as an infusion. Patients in group PA received propofol $9 \text{ mg.kg}^{-1}.\text{h}^{-1}$ as an infusion, decreasing to $6 \text{ mg.kg}^{-1}.\text{h}^{-1}$ after 15 min, together with alfentanil $10 \text{ }\mu\text{g.kg}^{-1}.\text{h}^{-1}$. Patients in groups IO and PO received isoflurane and propofol in the regimens described for groups IA and PA, but without alfentanil. Recovery was assessed by a blinded observer who recorded times to awakening (eye opening) and orientation (giving date of birth), and who repeated the psychomotor tests at 1, 3 and 5 h. Linear analogue scales of mood, nausea and pain were obtained and other side effects were noted in the succeeding 48 h. A matched control group of 25 females (who were not anaesthetised) underwent psychomotor testing on four occasions in order to assess the 'learning effect' of repeated recovery testing. The analysis of recovery tests did not assume a normal distribution. There were no significant differences in awakening or orientation times among the groups (median values IO: 4.92 and 6.06 min; PO: 3.88 and 5.11 min; IA: 6.90 and 7.98 min; PA: 6.24 and 7.30 min). There were also no differences among groups in respect of the results of psychomotor testing during recovery. There was significantly less nausea in group PO (6/32 patients) than in group IO (13/32 patients). There was also a significant difference between the performance of the control group compared with those of the study groups in most of the tests, and this emphasises the importance of including a control group in studies which involve recovery testing. We conclude that, after induction of anaesthesia with propofol in unpremedicated outpatients, maintenance of anaesthesia with nitrous oxide and either isoflurane or propofol, with or without alfentanil, provides reasonably rapid and reliable recovery and are equally acceptable to the patient.

Key words

Anaesthesia; outpatient.

Anaesthetics, intravenous; propofol.

Anaesthetics, inhalational; isoflurane.

The increasing role of outpatient surgery has emphasised the need for an anaesthetic technique characterised by smooth, reliable induction, rapid recovery and a low incidence of side effects [1]. Propofol has been recommended for use in outpatient anaesthesia [2, 3]. However, it has not been determined clearly whether the use of propofol for maintenance of anaesthesia confers any benefit in terms of speed of recovery compared with the use of an inhalational

agent such as isoflurane. Most studies which have compared maintenance of anaesthesia with propofol and isoflurane have been unblinded [3–7]. Those studies which have been blinded have been confined to brief (< 20 min) surgery [8, 9], or have used different induction agents in the propofol and isoflurane maintenance groups [10, 11]. We have undertaken a prospective, double-blind, controlled trial to determine the rate and quality of recovery from

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anaesthesia using propofol as an induction agent in all patients, and maintenance of anaesthesia with 67% nitrous oxide in oxygen and either propofol or isoflurane alone or with additional supplementation by an opioid analgesic. In addition, we introduced a matched control group who underwent tests in precisely the same manner as the study patients, but who did not undergo anaesthesia or surgery, in order to control for the effect of learning and time on the performance of recovery tests.

Patients and methods

Hospital ethics committee approval was obtained for the study, and informed consent was obtained from all the patients and from the control volunteers. Sixty-four female patients scheduled to undergo dental surgery were randomised prospectively into one of two equal groups to receive maintenance of anaesthesia with propofol (group PO) or isoflurane (group IO). In addition, 50 female patients scheduled to undergo gynaecological laparoscopy were randomised prospectively into one of two equal groups to receive maintenance of anaesthesia with propofol and alfentanil (group PA) or with isoflurane and alfentanil (group IA). All of the patients were ASA grade 1 or 2, had taken no central nervous system depressant drug within the last 14 days and were within 20% of their ideal body weight. Patients who had undergone anaesthesia within the previous month or who had any significant history of disorders of the cardiovascular, respiratory or neurological systems were not studied. In addition, a control group of 25 volunteers who did not undergo anaesthesia or surgery, but who fulfilled all other entry criteria, were studied.

None of the patients was premedicated. An intravenous cannula was inserted and an infusion of compound sodium lactate solution started at a rate of $3 \text{ ml.kg}^{-1}.\text{h}^{-1}$; this was continued until the patient was drinking again post-operatively. Anaesthesia was induced with propofol 2.5 mg.kg^{-1} in all four groups. Neuromuscular blockade was achieved with atracurium 0.3 mg.kg^{-1} , and the lungs were ventilated with 67% nitrous oxide in oxygen via a facemask. After tracheal intubation, mechanical ventilation was instituted using 67% nitrous oxide in oxygen in all patients. In group PO, propofol was infused at a rate of $9 \text{ mg.kg}^{-1}.\text{h}^{-1}$ for the first 15 min, and $6 \text{ mg.kg}^{-1}.\text{h}^{-1}$ thereafter. In group IO, isoflurane 1% was added to the inspired gas mixture. In group PA, the same propofol infusion regimen was employed as in group PO, but in addition, the patients received a bolus dose of alfentanil $10 \text{ }\mu\text{g.kg}^{-1}$ at induction and an infusion of alfentanil $10 \text{ }\mu\text{g.kg}^{-1}.\text{h}^{-1}$ thereafter. In group IA, this regimen of alfentanil administration was used in addition to supplementation of nitrous oxide with isoflurane 1%. Deviations from these regimens were at the discretion of the anaesthetist, and dependent upon clinical circumstances; all deviations were timed and recorded. At the end of surgery, residual neuromuscular block was reversed with neostigmine $40 \text{ }\mu\text{g.kg}^{-1}$, given with glycopyrronium $8 \text{ }\mu\text{g.kg}^{-1}$. When adequate neuromuscular function was present (assessed using train-of-four monitoring with a peripheral nerve stimulator), administration of anaesthetic agents was discontinued. Heart rate and blood pressure were measured immediately before induction of anaesthesia and at 5-min intervals thereafter. The electrocardiogram, oxygen saturation, end-tidal carbon dioxide concentration and expired

isoflurane concentration were measured continuously and recorded at 5-min intervals. A peripheral nerve stimulator was used to assess the degree of neuromuscular block.

The time to awaken (open eyes and/or squeeze hand on command) and the time to orientation (correct date of birth) were recorded by a blinded observer. The Steward score [12] was recorded for each patient on admission to the recovery ward and at 15 and 30 min after discontinuation of anaesthesia. Linear analogue scores for nausea were recorded at 30 min and 1, 3 and 5 h. The full battery of recovery tests (described below) was performed pre-operatively and at 1 and 3 h in patients who had undergone dental surgery, and also at 5 h in those who had undergone laparoscopy. Postoperative analgesia (ibuprofen 400 mg in groups PO and IO; papaveretum 0.3 mg.kg^{-1} , given with metoclopramide 10 mg, in groups PA and IA) was given if requested. All patients were given a questionnaire to complete at home; this was returned after 48 h.

Recovery testing

All the recovery tests were performed by an observer blinded to the anaesthetic technique employed. A computer package [13] was used to reduce observer bias. The following tests were used.

Pictorial memory test [14]. The patient was shown a card, which depicted nine objects, for 1 min. At the end of the testing session, which lasted approximately 15 min, the patient was asked how many objects she could recall from the card. A different card was used at each session.

Simple reaction time. The patient was instructed to press a button when four lights illuminated simultaneously on a panel. This test was performed three times at each session, and the mean result expressed in milliseconds.

Word memory test. Twenty words appeared on a screen over a period of one minute. At the end of this period, the patient wrote down as many words as she could recall in 2 min.

Salford tracker. A triangle on the screen was manipulated by the patient via a joy-stick, so as to track as accurately as possible the movement of another triangle which moved randomly across the screen. The sequence was repeated three times at each session and the results were expressed as the root mean square distance (mm) between the two triangles.

Linear analogue scales. Each patient used four linear analogue scales: anxious, happy, drowsy and energetic. On these scales, 0 represented the least anxiety (etc.) imaginable and 100 the most anxiety (etc.) imaginable. The scales appeared on the screen, and the patient moved the joy-stick from the mid-position to the position on the line which expressed her mood. One scale in each category was presented at each session. Two additional scales, one for nausea and one for pain, were evaluated at each session after operation. The control patients performed the tests on four occasions during the same day, and at the same times of day as the patients in the study groups.

Data are expressed as mean or median values with measures of variability expressed as SD or range. Data analysis was performed using the Wilcoxon Rank Sum test for continuous variables and Chi-square analysis or Fisher's Exact Probability test (for small numbers) for categorical variables. A *p* value of < 0.05 was considered to be statistically significant.

Table 1. Mean (SD) values for age, duration of surgery and times to awakening and orientation. Groups: IO, isoflurane only; PO, propofol only; IA, isoflurane/alfentanil; PA, propofol/alfentanil.

	IO	PO	IA	PA
Age; years	23.4 (4.8)	22.2 (3.5)	30.4 (4.1)	31.5 (5.5)
Duration; min	32.3 (3.9)	33.5 (9.4)	*40.2 (15.3)	34.5 (8.0)
Awakening; min	4.9 (2.4)	3.9 (2.0)	6.9 (3.8)	6.2 (3.4)
Orientation; min	6.1 (2.5)	5.1 (2.8)	8.0 (4.0)	7.3 (3.6)

* $p < 0.05$

Results

Data relating to age and duration of surgery are shown in Table 1; duration of surgery was significantly longer in group IA than in the other groups. Immediate recovery was assessed by times to awakening and orientation (Table 1) and the Steward score (Table 2; maximum score 6). There were no significant differences between group IO and group PO, or between group IA and group PA, but the addition of alfentanil added, on average, 2 min to the awakening and orientation times. All patients who had undergone dental surgery were discharged home after 3 h, except for one patient in group IO who returned to the operating theatre because of haemorrhage, and was unavailable for testing. Ten patients in group PA and seven in group IA were discharged before 5 h, and all patients in groups PA and IA were discharged home on the day of operation. The percentages of patients in each group who were unable to

complete the testing sessions because of nausea or drowsiness are shown in Table 3.

The results of the simple reaction time test are shown in Table 4. One hour after the end of anaesthesia, the reaction times were significantly slower than baseline in groups IO, PO and IA, but there were no differences from baseline 3 h after anaesthesia. In the control group, there was a significant improvement in performance at 1, 3 and 5 h, compared with baseline.

Results of the pictorial memory test are shown in Table 5. There was a significant deterioration from baseline in all groups at all times, except for group PA at 5 h. The control group also achieved lower scores at 1, 3 and 5 h. There was no significant difference from baseline in the word memory test in patients in groups PO and IO, but a variable decrease in performance in groups PA and IA, and a significant improvement in performance in the control group at 1, 3 and 5 h (Table 6) were demonstrated. There was a significant improvement in performance on the Salford tracker in group PO 3 h after the end of anaesthesia (Table 7). The performance of the control group improved significantly at 1, 3 and 5 h.

There were only minor differences among groups in respect of linear analogue scores for mood. Patients in group PO were significantly ($p < 0.05$) happier than those in group IO at 3 h, and patients in group PA were significantly ($p < 0.05$) happier than those in group IA at 1 h. Six patients in group PO complained of nausea after operation compared with 13 in group IO ($p < 0.05$); there was no difference between groups PA and IA in respect of nausea. Twelve patients in group PA experienced nausea, and 10 of these had received papaveretum after operation; another two patients received papaveretum but did not experience nausea. Twelve of the 15 patients in group IA who experienced nausea had received postoperative papaveretum; another four patients received papaveretum without subsequent nausea. There were no differences between groups PO and IO, or between groups PA and IA, in respect of analogue scores for pain.

Table 2. Median (range) of Steward scores.

	Admission to recovery	15 min after anaesthesia	30 min after anaesthesia
IO	5.3 (1-6)	6 (6)	6 (6)
PO	5.6 (1-6)	5.9 (5-6)	6 (5-6)
IA	5.9 (4-6)	6 (6)	6 (6)
PA	5.6 (1-6)	5.7 (5-6)	5.9 (5-6)

Table 3. Percentage of patients who were unable to perform psychomotor tests.

	PO	IO	PA	IA
1 h	9	16	20	36
3 h	0	*3	20	16
5 h	—	—	8	2

*Patient returned to operating theatre because of bleeding.

Table 4. Median (range) of simple reaction time (ms).

	PO	IO	PA	IA	Control
Pre-operative	295 (245-453)	295 (202-521)	317 (261-435)	317 (277-450)	312 (262-446)
1 h	*335 (248-503)	*330 (239-495)	325 (282-676)	*400 (295-548)	*282 (232-341)
3 h	320 (225-390)	309 (182-430)	320 (215-490)	342 (257-480)	*284 (203-325)
5 h	—	—	319 (233-407)	335 (248-377)	*289 (204-368)

* $p < 0.05$ compared with pre-operative or baseline value.

Table 5. Median (range) of pictorial memory scores (number of objects remembered).

	PO	IO	PA	IA	Control
Pre-operative	6 (3-8)	7 (3-9)	7 (0-9)	7 (4-9)	7(4-9)
1 h	*3 (0-7)	*4 (0-9)	*3.5 (1-7)	*3 (1-7)	*6 (4-9)
3 h	*3 (1-9)	*4 (0-9)	*5 (0-6)	*4 (0-8)	*5 (1-8)
5 h	—	—	6 (2-9)	*3 (0-8)	*5 (1-9)

*p < 0.05 compared with pre-operative or baseline value.

Table 6. Median (range) of word memory scores (number of words remembered).

	PO	IO	PA	IA	Control
Pre-operative	7 (1-11)	7.5 (1-12)	7 (2-12)	7 (3-11)	8 (3-12)
1 h	7 (2-12)	7 (3-17)	6.5 (3-11)	6 (2-13)	*9 (5-13)
3 h	6 (2-8)	6 (0-13)	*6 (0-8)	*5 (2-9)	*9 (5-13)
5 h	—	—	*6 (3-9)	6 (2-11)	*10 (3-13)

*p < 0.05 compared with pre-operative or baseline value.

There was a 75% response to the questionnaire. Of the respondents, 45% felt unwell on the journey home and 66.3% would have the same anaesthetic again. There were no significant differences among groups. Three patients volunteered the information that injection of propofol had been painful during induction, although this information was not sought.

There were 10 deviations from the protocol in group IO; the inspired isoflurane concentration was reduced in six patients and increased in three, and in one patient an additional dose of propofol 10 mg was given before tracheal intubation. In group PO, 10 patients received a total of 16 additional bolus doses of propofol 10 mg. In group PA, six patients each received one additional dose of propofol 10 mg. There were no deviations from the protocol in group IA.

Discussion

The speed, reliability and quality of recovery from anaesthesia is of paramount importance to the efficient running of the day-stay unit. Although many studies have demonstrated the advantages of propofol as an induction agent for outpatient anaesthesia [15, 16] the issue of maintenance of anaesthesia after propofol induction has been less widely

addressed. This study was designed to investigate whether intravenous maintenance of anaesthesia with propofol offered an advantage in terms of speed and quality of recovery compared with inhalational maintenance of anaesthesia with isoflurane after induction of anaesthesia with propofol in both groups.

Infusion rates of propofol and alfentanil were determined from the literature and from pilot studies. Numerous different manual and computerised infusion regimens for propofol have been described, depending on whether premedication, opioid or nitrous oxide are employed. Although equipotency between the isoflurane and propofol regimens is difficult to demonstrate, both regimens are employed commonly in the clinical situation. The propofol regimen has been described before [17, 18] and seems appropriate for use with nitrous oxide but no premedication. We undertook a pilot study using various bolus and infusion regimens of alfentanil in order to determine the most appropriate dose.

In an editorial 15 years ago, Epstein noted that research into recovery from anaesthesia had not been standardised despite the increasing popularity of outpatient surgery [19]. The situation seems little changed today [6, 8, 10, 20, 21] and the lack of standardised research makes the comparison of results from individual units difficult. We attempted to obtain as complete a picture of recovery as possible by using the Steward score, and wakening and orientation times to indicate the earliest aspects of recovery; the simple reaction time (preparation and motor), word memory (immediate recall), pictorial memory (delayed memory recall) and Salford tracker (complex psychomotor co-ordination) were used to cover a range of psychological and motor recovery. Linear analogues scales of mood and a questionnaire were used to determine mood effects, and patient perception and acceptability of the technique.

We believe that an important feature of our study was the presence of a control group. Most tests have a learning curve which may continue to show improvement in speed and accuracy of performance after several attempts until a

Table 7. Median (range) of Salford tracker scores (r.m.s. mm).

	PO	IO	PA	IA	Control
Pre-operative	16.6 (6.7-35.0)	16 (7.2-64.1)	17.3 (7.9-56.9)	18.5 (11.3-62.4)	14.7 (9.6-33.4)
1 h	15.5 (9.0-28.6)	15.6 (6.3-41.4)	19.1 (8.0-29.3)	21.1 (14.4-49.8)	*12.8 (9.0-30.1)
3 h	*14.4 (7.4-33.9)	13.8 (6.0-47.6)	16.3 (8.7-33.2)	20.2 (11.2-47.7)	*12.1 (8.3-29.0)
5 h	—	—	18.7 (7.9-54.3)	20.4 (10.6-46.6)	*11.9 (6.4-20.9)

*p < 0.05 compared with pre-operative or baseline value.

plateau is reached. In the context of a busy day-stay surgery unit it is not possible to achieve this plateau with patients and the control group therefore provides an invaluable indicator of a shortfall in performance which, in the patient groups, shows as a failure to improve on baseline scores.

The mean postoperative Steward scores did not demonstrate any difference between groups, nor did the addition of alfentanil adversely affect the mean scores. Two previous studies [22, 23] have shown alfentanil to have no effect on recovery time if given at induction or as preoperative boluses; although the awakening and orientation times were somewhat longer with the addition of alfentanil in our study, this difference was not statistically (or clinically) significant. It was noticeable that between 16% and 35% of patients who received alfentanil were unable to perform the tests at 1 and 3 h although this problem had been overcome by 5 h and all patients were discharged home on the same day. This suggests that if opioids are to be used in day-case surgery then their administration should be limited to the first half of the working day.

The simple reaction time scores showed no difference from baseline in any group 3 or 5 h after anaesthesia. Performance improved significantly in the control group at 1, 3 and 5 h indicating that an expected improvement in performance had not taken place in the patient groups. This affected all the treatment groups similarly. The pictorial memory test showed a deficit in all patients and the control group which seemed to be due to the subjects' becoming confused as to which picture they had seen on which occasion. It was not felt to be a highly discriminatory test. Word memory showed a decrease in performance in groups, PA and IA at 3 and 5 h whereas performance in the control group improved significantly. The lack of a significant difference from baseline at 1 and 3 h in the PO and IO groups is therefore indicative of a decrement in performance compared with control subjects. The Salford tracker also revealed an improvement in control performance without any improvement in patient groups. These psychomotor recovery tests therefore demonstrate no difference between propofol and isoflurane as maintenance agents, with or without alfentanil, but there was still evidence of a decrement in performance on three of the four tests at 3 h and 5 h respectively.

From its early use it was apparent that propofol was associated with a very low incidence of postoperative emesis [24]. Our study demonstrates an antiemetic effect of propofol compared with isoflurane maintenance when no opioids are given but this difference was lost when opioids were added to the technique. This confirms work by White and colleagues [25] which indicated that there were no differences in the incidences of nausea and vomiting between a group induced and maintained with propofol and a thiopentone/isoflurane group, if major surgery was performed and postoperative opioid administered.

No evaluation of an anaesthetic technique should be complete without some feedback from the patients. There was a high (75%) response rate to the postdischarge questionnaire. The number of patients who felt unwell during the journey home was high; the majority of complaints related to dizziness and nausea, with pain being quoted less frequently. The question 'would you have the same anaesthetic again?' needs to be interpreted with caution as the different groups all had the same induction experience with

propofol and there were a number of adverse comments on the pain associated with injection of propofol. This may have led some patients in the isoflurane as well as the propofol maintenance groups to reject the same anaesthetic again.

In summary, the only difference demonstrated between propofol and isoflurane as maintenance agents to supplement nitrous oxide for outpatient anaesthesia was a reduced incidence of nausea amongst patients who received propofol. The addition of opioid abolished this difference. Both anaesthetics provided reasonably rapid and reliable recovery from anaesthesia and were equally acceptable to the patients.

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Patient-controlled analgesia: a double-blind study in burn patients

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Summary

A double-blind controlled study was performed to assess the efficacy and safety of patient-controlled analgesia in burn patients. This method was compared with conventional analgesic therapy consisting of intermittent intravenous morphine injections. Twenty-four adult patients hospitalised for burn injuries participated in the study. The McGill Pain Questionnaire, visual analogue scales and verbal-numeric scales were administered at regular intervals to measure various components of the patients' pain experience, degree of pain relief, anxiety levels, adverse side effects and overall treatment efficacy. Although statistical significance was found in only one measure, the results suggested better pain control in patients who were administered morphine by patient-controlled analgesia as compared with intermittent injections. Analysis of the side effects showed no difference between the groups. The amount of morphine administered over the trial period was also similar for the two groups but considerable interpatient variability was seen. Although further research is needed to determine the conditions for optimal use of patient-controlled analgesia, it is concluded that it is a safe, effective and improved method for controlling pain in selected burn patients.

Key words

Burns; pain.

Analgesic administration; patient-controlled analgesia.

Patient-controlled analgesia (PCA) has gained increasing acceptance in the management of postsurgical pain [1–3]. However, little information exists on its suitability for other clinical conditions where the pain may be more severe and prolonged. Burn injuries cause one of the most intense types of pain, and lead to prolonged hospitalisation. Burn victims suffer not only the pain due to their burn injuries but also the pain associated with the multiple therapeutic procedures that must be carried out during the course of treatment (e.g. dressing changes, wound cleansing, physiotherapy, etc) [4–7]. Adequate pain control represents a major problem in these patients due to the wide variations in their analgesic requirements. The pain is extremely variable from one patient to the other, and it undergoes wide fluctuations over time in each patient. To be able to treat this pain effectively, flexibility in dosing is absolutely essential [4–7]. Because PCA allows the patients to adjust their pain medication according to their own needs, this technique could be suitable for burn pain.

Numerous clinical reports and studies have substantiated the efficacy and safety of PCA in nonburn patients. However, many of these studies were poorly controlled and showed methodological shortcomings [3]. Recent studies [8,9] have used more carefully controlled design to

compare PCA with traditional pain therapy, but two methodological problems still persist. Firstly, in all trials where PCA is compared with intermittent intramuscular injections of analgesics, the drug is administered by two different routes (i.e. intravenously vs intramuscularly). Because the intramuscular route is often associated with erratic drug absorption [10], it is possible that the observed difference between the two treatment modalities could be attributable, at least in part, to a difference in the route of administration. Secondly, none of the studies have used an adequate double-blind design to assess the placebo effect of PCA. Welchew [9] used a double-blind design to compare PCA with conventional pain therapy but his study was seriously criticised because different analgesic drugs (fentanyl vs morphine) and routes of administration (intravenously vs intramuscularly) were employed in his two groups [3].

In the literature, only two reports deal with the use of PCA in burn patients. Wermeling *et al.* [11] have described the successful use of PCA for a burn patient whose pain was refractory to conventional intravenous opioid therapy. Kinsella *et al.* [12] have reported results of a pilot study carried out in a small group of burn patients which suggested that PCA can be an effective method to alleviate

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pain for these patients. Further studies using adequate comparative designs are clearly needed to assess the therapeutic merit of PCA against other treatment methods for burn pain.

The present study was therefore undertaken to evaluate, in a double-blind controlled manner, the efficacy and safety of intravenous PCA in burn patients. The PCA mode was compared with traditional intravenous therapy consisting of intermittent injections administered as needed.

Methods

Subjects

Following approval by the hospital's Research and Ethics Committees, 24 adult patients hospitalised at the Burn Centre of the Hotel-Dieu Hospital of Montreal were recruited. Entry into the study was restricted to patients aged between 16 and 70 years, who were free of any significant pre-existing disease, and who had no history of substance abuse or chronic treatment with psychotropic or analgesic medication. Patients who were confused or who had injuries and complications too severe to allow normal communication were not studied, nor were those with an insufficient comprehension of the French or English language.

Experimental design

The study used a prospective, randomised, double-blind design to compare the efficacy of a PCA delivery system with conventional intermittent administration of analgesics. The intravenous route was used with both modes of administration to ensure that the study compared modes rather than routes of delivery. In order to carry out the trial in a double-blind manner, the two methods of treatment were employed simultaneously with all patients. Each patient was provided with a PCA pump and allowed intravenous injections every 3 h as needed. The PCA syringe and the vials for nurse-administered injections contained morphine (1 mg.ml^{-1}) or NaCl 0.9% solutions. Half of the subjects were randomly assigned to receive morphine from the PCA infuser and saline solution for the nurse-administered injections, while the other half received the opposite. The patient, the staff, and the research assistant were unaware of the group to which the patient was assigned. The syringes and vials were prepared and coded by the hospital's pharmacist. In case of an emergency, a copy of the code was kept in a sealed envelope on the ward.

Procedure

All participants were hospitalised within 24 h after the burn injury. On the second day following admission, the research assistant informed eligible patients about the study and requested their written consent to participate. A short structured interview was carried out to collect demographic and personal history data, and instructions in the use of the PCA pump were provided to each patient. The PCA device used was a Harvard PCA pump (Bard Canada Inc., Mississauga, Ontario, Canada). For the purpose of the present study, the control button was modified by the biomedical engineering department for easier use by burn patients with hand injuries.

The clinical trial started the morning after (third day postadmission) at a time when the patient next needed an

injection for pain. Using the solution contained in the vial coded by the pharmacist, the nurse administered an injection to the patient. The PCA pump was connected to an intravenous infusion line and programmed to administer a 3 ml loading dose. The pump was set to administer 1.0 ml bolus upon patient request, with a lockout interval of 6 min between doses.

After a review of PCA instructions, each patient was invited to activate the pump as often as necessary, and reminded about the availability of the nurse-administered injections. Dosage of these injections was determined on the basis of what the patient was receiving prior to the study (range 3 ml to 15 ml). The injections were offered at regular intervals and administered upon the patient's request or based on the nurse's judgement. If necessary, changes in dosage were made by the research assistant based on the patient's response. If the patient did not obtain acceptable levels of analgesia, the dosages of the PCA bolus and the nurse-administered injections were both adjusted in increments of 50% of the initial dose. If the patient was somnolent, the dosages were decreased by 25%. No sedatives, hypnotics, antihistaminics or analgesics other than morphine were allowed during the study period, which lasted 72 h.

Measurements

Patients' measurements. Every 4 h for the 72 h study period, the nurses asked the patients to rate the intensity of their present pain using a 10 cm visual analogue scale (VAS) which was adapted for easy use by burn patients with hand injuries [5]. If a patient was sleeping at the time of data collection, a missing value was assigned. At 24, 48, and 72 h after the start of the study, a second set of pain assessments was collected by a person not directly involved in the patients' care i.e. the research assistant. These measures included the McGill Pain Questionnaire [13] which was used to assess overall pain during the past 24 h; VAS scales were administered to measure pain intensity at its worst, at its least and overall during the previous 24 h; pain relief, anxiety and nausea ratings were also obtained from each patient with VAS scales. Upon completion of the study, the same type of scale was used by the patients to rate the overall efficacy of the analgesic treatment. When used to assess pain intensity, the anchor words of the VAS scales were 'no pain—worst pain imaginable'. For the anxiety and nausea scales, the anchors were 'not at all—extremely'; they were replaced by the expressions 'not at all—completely' when used to measure pain relief and overall treatment efficacy.

Nurses' measurements. Every 4 h, the treating nurse recorded respiratory rate, blood pressure and pulse rate. At the same time, she evaluated the degree of sedation on a 5-point scale, where 1 represented wide awake, 2 drowsy, 3 dozing intermittently, 4 sleeping frequently, 5 wakens only when aroused. Normal sleep was scored as a missing value. Any other adverse effects of the analgesic medication were also noted by the nurse. Finally, at the end of the work shift, each nurse was asked to provide a VAS rating for the overall efficacy of the analgesic treatment.

Medication intake and medical data. Once a day, the research assistant obtained a printout from the PCA pump. The number of PCA demands (successful and unsuccessful attempts) and nurse-administered injections was calculated

for each patient together with dosage adjustments. Upon completion of the study, the code to the solution contents was broken and the amount of morphine administered was calculated for each patient. Hospital charts were reviewed to retrieve relevant medical information. Severity of the burns was evaluated with the Lund and Browder's chart [14], and the total body surface area burned [TBSAB] was calculated by adding the percentages of first-, second- and third-degree burns.

Statistical analyses

A first set of analyses examined group comparability on demographic and medical variables, using *t*-tests (continuous data) and Chi-squared analyses (nominal data). Pain and sedation scores collected every 4 h were averaged over 24 h periods and analyses of variance (ANOVA) for repeated measures were used to test the group, time (days of treatment) and group-by-time interaction effects.

Comparable ANOVAs were performed on the outcome variables which were measured once a day and on the medication data cumulated over 24 h periods. For other measures, parametric (unpaired *t*-test) and nonparametric tests (Chi-squared analysis, Friedman ANOVA) were performed where applicable. The Yates' correction for continuity was applied to the Chi-squared test where appropriate. For all analyses, $p < 0.05$ was considered statistically significant.

Results

Group comparability

Table 1 shows that patients of the PCA and nurse-administered groups were comparable in age, sex, weight, and extent of burn injuries. The two groups also did not differ significantly with respect to the total amount of morphine (or its equivalent) they received on the 2 days prior to the study.

Pain, anxiety and overall efficacy measures

Figure 1 compares the pain scores of both study groups recorded at 4 h intervals with the VAS scale. The patients who were administered the morphine with conventional injections tended to report slightly more pain than those receiving morphine from the PCA infuser. When these VAS

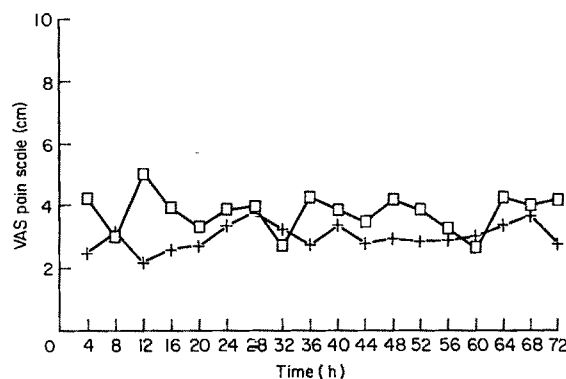


Fig. 1. Mean visual analogue pain scores of PCA (+) and nurse-administered (□) groups recorded at 4 h intervals for the duration of the study.

scores were averaged over 24 h periods, the mean values remained consistently higher in the nurse-administered group, but the difference failed to reach statistical significance. The ANOVA revealed that the pain scores were not varying as a function of group or time (days of treatment) and there was no significant group-by-time interaction.

Table 2 summarises the results obtained on the scales which were administered once a day. It can be seen that the group mean differences were all in the same direction, suggesting a trend for a better outcome with the PCA treatment. This was true for the data obtained on the McGill Pain Questionnaire and on the VAS scales measuring pain intensity (pain at its worst, its least, and overall during the past 24 h), pain relief and anxiety levels. However, statistical significance was not found on any of these measures. The ANOVAs revealed no significant group, time or group-by-time interaction effects.

The rating of the overall efficacy of the analgesic treatment showed the mean VAS score to be 8.4 (1.3) for the PCA group compared to 7.9 (1.9) for the nurse-administered group. This difference was not statistically significant. In contrast, overall efficacy ratings obtained from the nurses and averaged over the study period showed a significant difference between the two groups. The mean efficacy rating was superior for the group of patients who had the morphine administered with the PCA infuser ($\bar{x} = 7.3$ (1.0)) rather than intermittent injections ($\bar{x} = 6.1$ (1.3)) ($p < 0.02$).

Table 1. Demographic and medical characteristics of patients in each study group. Values are expressed as mean, (SD) and [range].

Variables	PCA group (n = 12)	Nurse-administered group (n = 2)	p
Age; years	38.0 (14.4) [21–63]	32.8 (9.3) [17–49]	ns
Sex: female/male	0/12	4/8	ns
Weight; kg	74.8 (10.8) [53.3–88.5]	72.3 (12.2) [51.6–83.1]	ns
Burn extent; % TBSAB	14.2 (10.9) [2.0–34.0]	14.4 (11.6) [1.5–41]	ns
Prestudy morphine intake; mg*	51.9 (28.4) [27–87]	84.0 (55.3) [31–225]	ns

* Total amount of morphine (or its equivalent in mg according to Jaffe and Martin [15]) administered on the 2 days prior to the study.

PCA, patient-controlled analgesia; % TBSAB, percent of total body surface area burned.

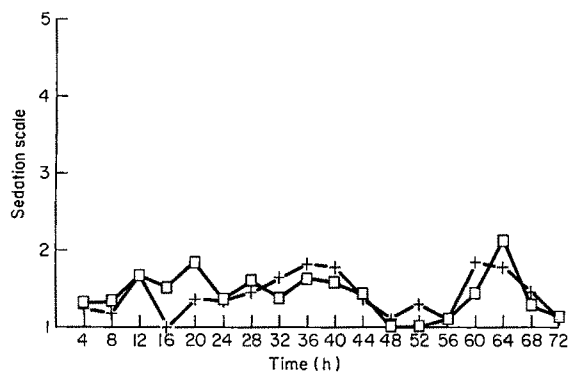


Fig. 2. Mean sedation ratings of the two study groups recorded at 4 h intervals for 72 h. +, PCA; □, nurse-administered.

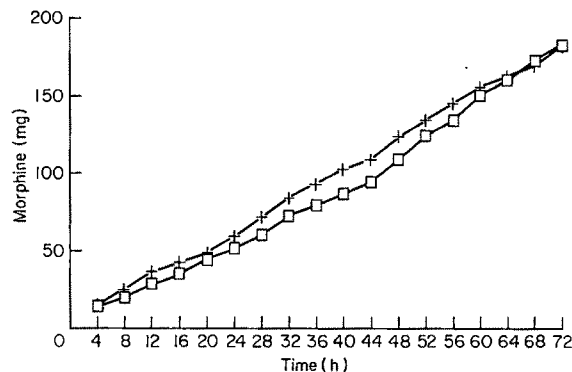


Fig. 3. Cumulative morphine intake (in mg) in PCA (+) and nurse-administered (□) groups during the 72 h of the study.

Side-effect profile

Figure 2 shows the mean sedation ratings for the PCA and nurse-administered groups every 4 h for the duration of the study. The ANOVA revealed no significant group, time (days) or group-by-time interaction effects on the sedation scores. Patients of the nurse-administered group were recorded wide awake on 76% of the evaluations, and drowsy, dozing or sleepy on 24% of the evaluations (total observations = 179). In the PCA group, the percentages were 77% and 23% respectively (total observations = 188). No incident of respiratory depression or hypotension was observed with either method of treatment during the study.

Little or no nausea was reported by the majority of patients treated with the PCA or nurse-administered methods. The mean VAS score of the PCA patients was 0.8 (1.5) for the 3 days of the study, compared to 0.9 (1.7) in the other group. The ANOVA analysis confirmed that the VAS nausea scores did not differ significantly between the groups across the study days.

Medication intake

Cumulative morphine intake over the whole study period is displayed in Figure 3 for the PCA and nurse-administered

groups. Statistical analysis revealed no significant difference in daily drug consumption as a function of group and/or study days. The mean total doses of morphine for the 3 days were 182.9 (94.8) mg for the PCA group and 183.6 (110.1) for the nurse-administered group with considerable interpatient variation in both groups: 70 mg to 321 mg for the PCA group, and 74 mg to 443 mg for the nurse-administered group. No significant correlation was found between morphine use and extent of burn injuries.

Further analysis of the medication data revealed a significant difference in the number of dosage changes required over the study period in each group ($p < 0.04$). In the nurse-administered group, all patients (12/12) required one change or more. In the PCA group, 58% (7/12) of the patients required an increase or decrease in medication.

A last series of analyses (nonparametric Friedman ANOVAs) tested whether patients were kept blinded to the mode of therapy across the 3 days of the trial. Results of the analyses tended to support 'blinding' of the study. In the nurse-administered group, no significant change was observed in the number of PCA demands from day 1 to day 3 (median values (MD) = 181.0, 182.5, 207.5). In the PCA group, the number of intravenous supplements tended to decrease over time but the difference was not statistically significant for the 3 days of the study (Day 1, MD = 3.3; Day 2, MD = 2.0; Day 3, MD = 1.5).

Table 2. Comparison of the mean (SD) scores obtained on pain measurements, pain relief and anxiety scales for patients of the PCA and nurse-administered groups. Group ratings are averaged for the 3 days of the study period; the p value represents the ANOVA probability associated with group effect result.

	PCA group (n = 12)	Nurse-administered group (n = 12)	p
Pain scales			
McGill Pain Questionnaire*	29.0 (17.6)	39.4 (13.7)	0.09
Visual analogue scales; cm			
Pain at its worst	6.8 (2.0)	7.7 (1.1)	ns
Pain at its least	1.0 (0.7)	1.4 (0.6)	ns
Overall pain	3.7 (1.6)	4.3 (1.3)	ns
Pain relief scale (VAS); cm	7.2 (0.8)	6.6 (1.4)	ns
Anxiety scale (VAS); cm	3.5 (2.0)	4.3 (1.9)	ns

* McGill Pain Questionnaire data on day 1, 2 or 3 were missing for two patients in the PCA group and one in the nurse-administered group.

Discussion

This trial used a double-blind controlled design to evaluate and compare the efficacy of PCA against standard analgesic regimen for controlling pain in burn patients. Results obtained on the various outcome measures were all suggestive of a trend for superior pain control with the PCA technique. Statistical significance was not found, however, except on one measure (nurses' efficacy ratings).

Several factors can explain the lack of statistical significance. First, it may have been due to the small number of patients evaluated and the large interpatient variability in pain perception and response. A greater number of subjects in each group would, perhaps, have increased the likelihood of statistically significant results.

Another factor to take into account when interpreting these results is the double-blind nature of the study. Several authors [9,16] argue that PCA pumps provide a substantial placebo effect. Others [2,8] maintain that a key factor in PCA success is the patients' satisfaction resulting from the feeling of being in control. In other words, success of PCA therapy may not only lie in the actual superiority of the technique but may involve other factors. In the present study, all patients were provided with a PCA pump and were, therefore, given the opportunity to realise some of the associated effects of PCA (e.g. sense of control). Exposing patients of the nurse-administered group to these effects may have contributed to obscuring group difference. In the light of these considerations, it is tempting to speculate that the observed pattern of results could argue in favour of the superiority of PCA therapy, even if statistical significance was not found.

However, another factor which could have contributed to the present findings may be related to an inherent limitation of the PCA method. To be effective, the method requires the patient to be both awake and responsive in order to maintain adequate drug delivery [2,17]. Thus, reductions in PCA dosings during sleep hours may result in subtherapeutic plasma concentrations. When the patient awakens, pain levels experienced at that time can be comparable to those observed with conventional injections. Furthermore, patients may reduce the efficacy of the PCA method by not adequately medicating themselves before painful treatment or movement [2,17]. It has been suggested that the addition of a continuous background infusion could improve the uniformity of analgesia provided by PCA [1,2,17]. Although this combined method was shown to be an effective pain-control strategy [18,19], inconsistent results were obtained in some recent studies [17,20,21] which tried to demonstrate the therapeutic superiority of the combined method over the standard PCA approach. Conditions for optimal use of PCA in combination with a basal infusion still need to be defined and more research must be carried out to assess the therapeutic merit of this method. In future studies with burn patients, treatment effects should be examined separately: for the pain felt at rest and at times of therapeutic procedures. It is possible that a PCA approach provides excellent pain control when the patient is resting but may not suffice for the intense pain levels felt during therapeutic procedures. On these occasions, an intravenous bolus administered prior to the procedure may be necessary to supplement the PCA regimen.

Contrary to earlier claims [22,23], the results of the

present study failed to confirm that PCA-treated patients require less opioids than patients receiving conventional intermittent injections. Previous studies used different routes to administer the medication, the intravenous route being favoured for the PCA mode and the intramuscular route for intermittent injections. It is well known that opioid drugs, when administered intramuscularly, are often poorly or erratically absorbed [10]. As a result, high doses may be needed to achieve levels of analgesia comparable to those observed with intravenous PCA therapy. In the present study, the intravenous route was employed in both analgesic regimens to ensure that methods were compared rather than routes of administration. The results showed comparable levels of drug intake in the two treatment groups, a finding consistent with more recent studies [8,24,25].

Considerable variability was observed in the amount of medication the patients required. Morphine consumption varied four- to five-fold in both groups, further confirming the marked variation among patients in regard to analgesic requirements, along with the need for highly individualised treatment strategies [10,22]. Dosage adjustment results also illustrate the difficulty for a prescribing physician to find the right dose for each patient. All patients who were administered morphine by intermittent injections required one or more changes of dosage during the study period. Some dosage adjustments were also required in the PCA group, but the number was significantly lower. As pointed out by many authors [1-3], PCA therapy has the advantage of accommodating a wider range of analgesic requirements.

Consistent with some previous observations [8,25], the present study revealed comparable side effect profiles for PCA and conventional treatment. Furthermore, respiratory depression was not observed in any patient and no evidence of opioid abuse was noted, further confirming that PCA is a safe method for controlling pain.

In the light of the results of the present study, it is concluded that PCA is a safe, effective and improved method for controlling burn pain. It can offer good pain relief and individualised treatment in burn patients who are stable, alert and able to understand how to use the system. However, continued research is needed to overcome certain limitations of PCA. Additional studies using a double-blind controlled design must also be performed to test the efficacy of PCA against conventional analgesic therapy so as to replicate the results of the present study in other clinical settings.

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The absence of teratogenic effects of some analgesics used in anaesthesia

Additional evidence from a mouse model

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Summary

The possibility exists that agents used in anaesthesia may have adverse teratogenic effects on staff, patients, and developing fetuses. It has been shown that a range of neurotropic drugs, when injected into pregnant mice on the 9th day of gestation, produce a characteristic group of central nervous system malformations in their fetuses. We have studied the possible teratogenicity of pethidine, fentanyl, phenoperidine and lignocaine when tested in this way and conclude that they appear to have less effect than other neurotropic drugs previously tested.

Key words

Toxicity; teratogenicity.

Analgesics; opioids.

Anaesthetics, local; lignocaine.

The fact that the administration of drugs to women in early pregnancy can result in developmental defects in the fetus was highlighted by the thalidomide disasters of the 1960s [1]. The possibility that agents used in anaesthesia can produce similar effects has given cause for concern.

The commonly used inhalational agents have been extensively investigated. Their teratogenic effects in animals have been studied and evidence of susceptibility in man sought. Attention has been directed both to chronic exposure of operating theatre personnel to trace concentrations of anaesthetic gases and vapours and to the effects on the fetus of general anaesthesia administered to women in early pregnancy.

It has been shown that some neurotropic drugs when administered to mice on the 9th day of pregnancy produce a characteristic group of central nervous system malformations in the fetuses [2,3]. This neurotropic syndrome of malformations consists of exencephaly, craniorachischisis, cervical and thoracolumbar myeloschisis, hydrocephalic dilatation of the fourth ventricle, Z-shaped kinking of the spinal cord and lumbar hydromyelia.

Drugs which have been shown to produce these effects include tricyclic antidepressants, phenothiazines, benzodiazepines [3], lithium [4], butyrophenones [5], and a range of opioids, both naturally occurring and synthetic [2,3,5].

In this paper we report an investigation of the effects on

fetal murine CNS development of pethidine, phenoperidine, fentanyl and lignocaine.

Methods

In this study the JBT/Jd strain of laboratory mouse inbred for 95 generations by brother–sister matings was used. The experimental animals were accommodated in the animal house under normal day/night illumination conditions. Two or three females 2–3 months old were kept with one male of similar age between 1000 h and 1600 h. During this time, at 2 h intervals, the females were examined for the presence of copulation plugs. By convention the 24 h period following the finding of copulation plugs was considered to be the first day of pregnancy.

The drugs tested were administered in sterile physiological saline solutions and the concentration of each was adjusted to give an injected volume of 0.02 ml.g⁻¹. The drugs were given intraperitoneally, under light ether anaesthesia, 1 h into the 9th day of gestation. Four days later i.e. on the 13th day of gestation, the animals were killed by cervical dislocation. The entire uteri were excised and fixed *in toto* using a 5% solution of trichloroacetic acid containing 1.3% lanthanum acetate. After a minimum of 3 h the fetuses were dissected out of the uteri, macroscopically examined under the dissecting microscope, and any gross malformations recorded. They were then stored in a fresh fixative

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solution and histological examination carried out where the nature and extent of any malformations was in doubt.

Retardation of development is one of the symptoms of embryotoxicity and was evaluated with the help of data from our own reference collection of embryos and fetuses together with findings of Gruneberg [6] and Theiler [7]. Experimental fetuses were recorded as retarded if in comparison with these three reference standards their external features indicated 1 to 2.5 days retardation of development.

For control purposes, additional pregnant females were weighed, lightly etherised and injected with the appropriate volume of 0.9% saline solution at 1 h into the 9th day of gestation. After a further 4 days these animals were killed and their litters fixed for use as general and histological controls.

Preliminary experiments were performed to determine the median adult lethal doses (LD_{50}) by means of graded dose-lethal response tests. For this purpose nonpregnant females were treated by single intraperitoneal injection. Teratogenic activity was then investigated using graded dose-response tests with doses lower than the relevant LD_{50} .

Results

The LD_{50} for pethidine was estimated at 110 mg.kg^{-1} , for phenoperidine at 34 mg.kg^{-1} , for fentanyl at 16 mg.kg^{-1}

Table 1. Graded teratogenic dose-response relationships of pethidine. Treatment by intraperitoneal injection under light ether anaesthesia 1 h into the 9th gestational day.

Dose (mg.kg^{-1})	Number of fetuses (13th gestation day)					
	Total	Dead	Normal		Retarded +/- CNS anomalies	
			<i>n</i>	%	<i>n</i>	%
Control	65	1	64	100	—	—
80	24	6	16	88.8	2	11.2
81	8	—	8	100	—	—
82	35	2	31	93.9	2	6.1
83	21	—	15	71.4	6	28.6
85	89	6	81	97.6	2	2.4
90	16	1	14	93.3	1	6.7
100	15	—	13	86.7	2	13.3

Table 2. Graded teratogenic dose-response relationships of phenoperidine. Treatment by intraperitoneal injection under light ether anaesthesia 1 h into the 9th gestational day.

Dose (mg.kg^{-1})	Number of fetuses (13th gestation day)					
	Total	Dead	Normal		Retarded +/- CNS anomalies	
			<i>n</i>	%	<i>n</i>	%
Control	74	2	72	100	—	—
18	16	—	8	50	8	50
22	58	4	48	88.9	6	11.1
23	24	—	19	79.2	5	20.8
25	11	2	7	77.8	2	22.2
28	13	1	12	100	—	—
32	19	3	12	75	4	25

Table 3. Graded teratogenic dose-response relationships of fentanyl. Treatment by intraperitoneal injection under light ether anaesthesia 1 h into the 9th gestational day.

Dose (mg.kg^{-1})	Number of fetuses (13th gestation day)					
	Total	Dead	Normal		Retarded +/- CNS anomalies	
			<i>n</i>	%	<i>n</i>	%
Control	101	—	100	99	1	1
12	11	—	8	72.7	3	27.3
14.5	48	8	25	62.5	15	37.5
15	19	4	12	80	3	20
16	262	29	165	70.8	68	29.2

Table 4. Graded teratogenic dose-response relationships of lignocaine. Treatment by intraperitoneal injection under light ether anaesthesia 1 h into the 9th gestational day.

Dose (mg.kg^{-1})	Number of fetuses (13th gestation day)					
	Total	Dead	Normal		Retarded +/- CNS anomalies	
			<i>n</i>	%	<i>n</i>	%
Control	80	1	79	100	—	—
32	20	1	18	94.7	1	5.3
40	9	—	9	100	—	—
50	6	—	3	50	3	50
55	6	—	3	50	3	50
58	4	1	1	33.3	2	66.6
60	47	2	40	88.8	5	11.2
66	26	3	18	78.3	5	21.7
70	20	2	16	88.8	2	11.2

and for lignocaine at 85 mg.kg^{-1} . The graded teratogenic dose-response relationships for pethidine, phenoperidine, and fentanyl are shown in Tables 1, 2 and 3, respectively. Pethidine is virtually free from teratogenic activity, only two fetuses showed exencephaly. Five fetuses were retarded and the remainder showed the minor abnormalities of spinal cord kinking and fourth ventricle dilatation only. The results with phenoperidine were similar with more fetuses showing growth retardation. The effect of fentanyl was slightly more pronounced with a similar range of abnormalities. In the case of lignocaine, dilatation of the fourth ventricle was the most frequent anomaly (Table 4).

Discussion

There is evidence that the commonly used inhalational anaesthetic agents can be teratogenic when administered to pregnant animals. The exposure of rats to nitrous oxide in concentrations of 50–75% for periods of 24 h or more during various stages of pregnancy has been shown to produce increased fetal resorption, diminution in size with a high incidence of malformations, particularly skeletal, in survivors [8,9]. The situation regarding the volatile agents is unclear. Basford and Fink [10] have shown that the inhalation of 0.8% halothane by rats for 12 h on one of days 6–10 of gestation, produces skeletal abnormalities similar to those caused by nitrous oxide. In contrast, Mazze

and coworkers [11] did not observe any effect with 0.8% halothane, 1.08% isoflurane, or 1.65% enflurane when given for 6 h consecutively on days 14–16 of gestation.

Whereas the possible teratogenic effects of inhalational agents have been extensively investigated, other drugs used during anaesthesia have received less attention. Nevertheless, morphine has been shown to produce diminished fetal growth, skeletal abnormalities and exencephaly when injected subcutaneously into mice on the 8th or 9th day of gestation [12] although the doses required are high, being 100–500 mg.kg⁻¹ relative to the LD₅₀ of nonpregnant mice of 525 mg.kg⁻¹. The continuous administration of similar amounts using osmotic minipumps implanted on days 7 to 10 of gestation produced similar effects [13]. The work of Geber and Schramm [14] in hamsters has shown that the teratogenic effect of opioids is not confined to mice and Jurand [3] has demonstrated that their susceptibility is not confined to opioids, other neurotropic drugs having similar teratogenic effects. Lignocaine has produced neural tube defects in mice embryos maintained *in vitro* [15]. The Committee on Safety of Medicines has recently drawn attention to the fact that noscapine, a constituent of papaveretum, has been shown to induce polyploidy in mammalian cell lines *in vitro* [16].

There has been concern that the administration of anaesthetics to pregnant women may have adverse effects on their fetuses. Retrospective surveys have been carried out of women who had undergone anaesthesia and of the anaesthetic histories of women producing offspring with congenital malformations, but no association between congenital malformations and anaesthesia has been shown [17–19].

A large retrospective survey by Duncan and colleagues [20] using health insurance data linked to a congenital anomaly register in Manitoba matched 2565 pregnant women undergoing operation with a similar number of controls. The incidence of congenital malformations was similar (1.68% and 1.52% respectively). Mazze and Kallen [21], in a Swedish survey of 2252 nonobstetric operations during the first trimester, showed a 5% incidence of congenital malformations of which only 1.89% were classified as important. This did not differ significantly from the incidence expected in the normal population.

The work reported here shows that the structurally related opioids—pethidine, phenoperidine and fentanyl—are virtually free from CNS teratogenicity in the mouse model used. A similar absence of teratogenicity has been demonstrated in Sprague–Dawley rats subjected to high doses of fentanyl delivered by implanted osmotic minipumps over a period of 2 weeks before and during pregnancy [22]. Furthermore, the administration of fentanyl by minipump from day 7 of pregnancy did not enhance the effect of nitrous oxide administered for 24 hours on day 9 [8].

The significance of this experimental work to the problem of the administration of anaesthetic agents to pregnant women must remain speculative. Interspecies variations are notorious and although the mouse placenta is anatomically and physiologically of the same haemochorial type as human placenta [23] direct extrapolation of the findings to man is unwarranted. However, the possibility remains that the administration of drugs at critical times during early embryogenesis can induce fetal malfor-

mations. The evidence presented here suggests that the phenyl-piperidine group of opioids are less likely to produce these effects than other opioids and that lignocaine is a safe local anaesthetic agent to use in early pregnancy.

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Admixture of propofol and alfentanil

Use for intravenous sedation and analgesia during transvaginal oocyte retrieval

E. SHERRY

Summary

An admixture of propofol and alfentanil provides adequate sedation and analgesia during transvaginal oocyte retrieval in the absence of a paracervical block. In 100 patients the technique provided haemodynamic stability, sedation which was easily controlled, rapid recovery and universal patient acceptance.

Key words

Anaesthetics, intravenous; propofol.

Analgesics; alfentanil.

The continuous infusion of propofol is a successful method of sedating patients in a variety of clinical settings. Initial experience in this unit demonstrated that propofol alone provided inadequate sedation for transvaginal oocyte retrieval in the absence of a paracervical block. Although it may provide some analgesia [1,2] additional analgesia in the form of premedication, intermittent doses or continuous infusion of an analgesic drug was required. Premedication is usually undesirable in day-case patients. Intermittent dosage is unsatisfactory as it is difficult to anticipate painful stimuli during the surgical procedure and easy to give an excessive dose.

An admixture of 50 ml propofol (10 mg.ml⁻¹) with 4 ml alfentanil (0.5 mg.ml⁻¹) has been used successfully in over 400 patients during the past 3 years to provide sedation and analgesia during transvaginal oocyte retrieval. This mixture contains propofol 9.3 mg.ml⁻¹ and alfentanil 37 µg.ml⁻¹. Admixture of propofol and alfentanil in the ratio 1:1, 20:1 and 50:1 in the same syringe are stable over a 24 h period when stored in 20 ml Beckton Dickinson Plastipax or Gillette Sabre syringes; the 1:1 mixture is unstable when continuously agitated (personal communication Janssen Pharmaceutical Ltd).

Methods

Data were collected prospectively on 100 consecutive women presenting for oocyte retrieval. All were day-case patients of ASA class 1 or 2 and unpremedicated following an overnight fast. An 18-gauge cannula was inserted into a

forearm vein in the operating theatre and a 5% dextrose infusion commenced. Continuous ECG and peripheral oxygen saturation (SpO₂) were monitored, arterial blood pressure (Critikon Dinamap) recorded and sedation level assessed at 5 min intervals, the latter using a five-point sedation score (Table 1) [3].

An infusion of the admixed propofol and alfentanil was commenced using a calibrated Ohmeda 9000 syringe pump at 2 ml.kg⁻¹.h⁻¹. Verbal contact was maintained with the patient throughout the induction period. When the patient became drowsy (ptosis or slurred speech) the infusion was decreased to 1 ml.kg⁻¹.h⁻¹ and oxygen-enriched air administered through a Hudson mask. The rate of infusion during the remainder of the procedure was adjusted according to the clinical response. The aim of sedation was to keep patients asleep but rousable to mild physical stimulation. Supplemental doses of the mixture were administered if patients moved or complained of pain.

When the ovarian follicles were visualised on the ultrasound scanner a needle with a very sharp tip was inserted through the needle guide on the vaginal probe. The needle passed through the vaginal wall into the follicle and aspiration commenced. There may be up to 30 follicles to be punctured following pre-operative ovarian stimulation with buserelin and follicle stimulating hormone. Oocyte retrieval is carried out under subdued lighting because of the inhibitory effect of fluorescent light on oocyte function. The patient remained in the lithotomy position with the vaginal probe in place for the duration of the procedure.

The following variables were recorded: the presence of

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Table 1. Sedation score.

1) Fully awake and orientated
2) Drowsy
3) Eyes closed but rousable to command
4) Eyes closed but rousable to mild physical stimulation (ear lobe tug)
5) Eyes closed but unrousable to mild physical stimulation

ain or discomfort at the start of the continuous infusion and the response following intravenous injection of 1 ml of 1% lignocaine, respiratory obstruction or apnoea, additional doses of the mixture administered and the time taken from end of infusion for patients to recall their date of birth. Patients were assessed for drowsiness, nausea and vomiting 20 min and 4 h following the operation and asked about the acceptability of the procedure.

Changes in arterial blood pressure and heart rate were analysed by ANOVA with the Scheffe multiple comparison procedure [4] and $p < 0.05$ was considered significant.

Results

The patients were of mean age 33 years (range 25 to 46 years) and mean weight 62 kg (range 36 to 100 kg). Drug dosages and other details are presented in Table 2. The amount of propofol and alfentanil used for maintenance excludes the induction dose but includes any additional doses administered after induction.

Induction

Thirty patients complained of local pain or discomfort on commencing the infusion but this responded rapidly to intravenous lignocaine (1 ml 1%). Respiratory obstruction, apnoea or excitatory phenomena were not observed in any patient during the induction period. Following induction there were significant decreases in heart rate (10%), systolic (5%) and diastolic (13%) arterial blood pressures (Fig. 1).

Maintenance

The mean interval between commencement of the maintenance infusion and insertion of the vaginal probe was

Table 2. Dosages and duration of infusions.

	Mean (SD)	Range
Induction		
Duration; min	3.6 (0.76)	2.4–7.2
Propofol; mg.kg^{-1}	1.1 (0.24)	0.74–2.2
Alfentanil $\mu\text{g.kg}^{-1}$	4.5 (0.9)	2.99–8.88
Start of infusion to surgery; min	9.0 (3.7)	5–20
Maintenance		
Duration of surgery; min	53.0 (14.3)	20–90
Propofol; $\text{mg.kg}^{-1}.\text{h}^{-1}$	6.8 (1.7)	3.8–13.5
Alfentanil; $\mu\text{g.kg}^{-1}.\text{h}^{-1}$	27.1 (6.8)	15.2–54.3
Recovery		
End of infusion to eyes open and recall date of birth; min	4.8 (2.9)	0–14

9 min. This represented surgical time and included gowning, preparation of the probe and adjustments of the ultrasound scanner. Sixty patients required additional doses of the mixture during the operation to increase the level of sedation. Twenty-one patients developed respiratory obstruction as evidenced by noisy breathing but there were no periods when the SpO_2 fell below 90% and no episodes of apnoea. There was no relation between respiratory obstruction and the administration of additional doses of the mixture. All cases of obstruction resolved within 5 min following support of the airway and reduction in the rate of infusion. Although 15% of patients reached stage 5 sedation during the procedure, none required the insertion of an airway. No patients had to receive a general anaesthetic. After induction, the heart rate and systolic and diastolic arterial blood pressures remained stable. The distribution of sedation scores is presented in Table 3.

Recovery

Recovery was rapid with patients remarking on their clear-headedness. No patient requested postoperative analgesia or complained of nausea or vomiting. Typically, patients were back in their room within 1 h, able to eat a light snack, and fit for discharge 4 h after leaving the operating theatre. No patient had any recall of the transvaginal puncture although 14 could remember either background noise or conversation with the anaesthetist during the

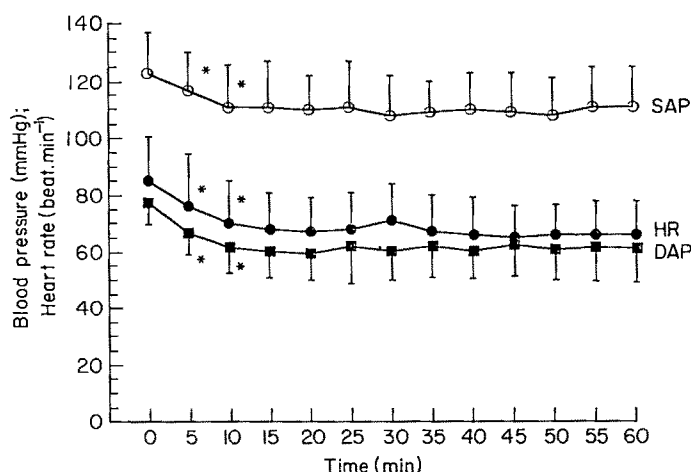


Fig. 1. Effect of continuous infusion of an admixture of propofol and alfentanil on mean (SD) systolic arterial pressure (SAP), heart rate (HR) and diastolic arterial pressure (DAP). * $p < 0.05$, significant change from preceding measurement.

Table 3. Distribution of sedation scores ($n = 100$).

Sedation score	Frequency (%)
1	11
2	12
3	22
4	40
5	15

procedure; none of them found this distressing and none recalled any pain. All the patients said that they would be willing to undergo *in vitro* fertilization (IVF) with this sedation technique again.

Discussion

An admixture of alfentanil and propofol provided adequate sedation and analgesia for transvaginal oocyte retrieval in the absence of a paracervical block. The mean dose of propofol used for induction was similar to that previously reported but the rate of administration was slower and may explain the absence of side effects during induction. The addition of alfentanil was uncomplicated and the dose used for both induction and maintenance was less than that used for general anaesthesia. The mean dose of propofol infused for maintenance ($6.8 \text{ mg.kg}^{-1}.\text{h}^{-1}$) is higher than that reported for sedation during regional anaesthesia but less than the ED_{50} reported for loss of the eyelash reflex in young unpremedicated patients [5].

The 30% incidence of pain or discomfort on injection of the admixture even when freshly prepared from propofol stored at 4°C and injected slowly into a forearm vein is similar to that reported by other workers [6].

Patients had sedation scores of three and four for 62% of the operation time. A sedation score of five represented 'anaesthesia' rather than sedation and, although 'safe' in an anaesthetic sense, is undesirable.

Oocyte retrieval under the guidance of a vaginal transducer is a successful surgical technique. Feichtinger and colleagues [7] reported the surgical technique performed on 61 women who had oocyte retrieval following premedication with 2.5 mg haloperidol, 2 mg flunitrazepam, 30 mg pentazocine and 0.25 mg atropine. A regional block was not used and patients were discharged home 1 h after surgery. No data were provided on the quality of sedation or recovery characteristics. Hammarberg and colleagues [8] reported the same technique performed on 51 Swedish women following a combination of premedication with 75 mg pethidine and the application of a paracervical block with 10 ml of 1% lignocaine; 28% of these patients required peroperative midazolam and 44% described pain during the procedure.

Subanaesthetic infusions of propofol provide excellent sedation during regional anaesthesia [9], endoscopy [10], lithotripsy [11] and in the intensive care unit [12]. The depth of sedation is easily altered by adjusting the infusion rate and the technique has a low incidence of cardiorespiratory side effects. Recovery, as judged by the ability to open the eyes and recall date of birth, is significantly faster after an infusion of propofol than an infusion of midazolam [3].

Alfentanil alone has been used to provide sedation and analgesia for extracorporeal shock wave lithotripsy of gallbladder stones [13] and during cardiac catheterisation [14].

There are no data on recovery characteristics following this technique and respiratory depression has been reported during and after continuous infusions of alfentanil [15].

None of the patients in this series received a regional block or routine intravenous lignocaine because of the risk of inhibition of cell growth in the laboratory by local anaesthetic [16]. The concentration of propofol found in follicular fluid after a single anaesthetic induction dose has been shown not to affect the embryo cleavage rate [17]. The success rates for IVF in women under 40 years in this unit (32% pregnancy rate and 31% birth rate) compare favourably with those of other centres and suggest that this combination has no important effect on embryo development.

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Anaesthesia for bullectomy

Use of propofol, high frequency jet ventilation and extradural blockade

A. F. KAN AND T. E. OH

Summary

A patient with a single large bulla occupying 50% of the right hemithorax was anaesthetised successfully with a combination of techniques not previously described. The technique consisted of extradural analgesia and intravenous infusion of propofol, while ventilation was maintained with high frequency jet ventilation through a single lumen tracheal tube. The advantages of this technique are a reduced risk of barotrauma, good operating conditions and good analgesia.

Key words

Anaesthesia; thoracic.

Anaesthetic techniques regional; epidural.

Anaesthetics, intravenous; propofol.

Giant lung bullae in patients who need surgery for the underlying condition or for other extrathoracic procedures pose problems for general anaesthesia [1–3]. Moreover, these patients frequently have underlying chronic lung diseases which further increase the risk of peri-operative morbidity and mortality. This case report presents a technique which minimises the potential complications associated with general anaesthesia in such patients.

Case history

A 61-year-old man with a 30-year history of heavy smoking was referred to the hospital for further management of a giant lung bulla. He complained of intermittent dysphagia and a recently decreased effort tolerance, both of which were symptoms consistent with an enlarging bulla. He had chronic obstructive pulmonary disease which was treated with beclomethasone, ipratropium and salbutamol inhalers supplemented by oral terbutaline, theophylline and prednisolone. The pre-operative chest X ray (Fig. 1) showed that the bulla occupied more than 50% of the right hemithorax. His electrocardiogram (ECG) was essentially normal, but lung function tests showed a severe obstructive/restrictive defect (Table 1). Arterial blood gases (Table 2) showed some hypoxaemia in room air, with normocapnia. Since the patient was symptomatic and there was radiological evidence of compression of surrounding lung tissue, surgical bullectomy was indicated [4].

No premedication was given. The nebulised drugs were given shortly before surgery with intravenous hydrocorti-

sone 100 mg. In the induction room, after venous access was established, an epidural catheter was inserted at the T₅₋₆ interspace, with the patient awake in the left lateral position.

After a pre-oxygenation period of 3 min, intravenous propofol 80 mg was given, followed by suxamethonium 75 mg to facilitate tracheal intubation with a size 9.0 Mallinckrodt 'Hi-Lo Jet' cuffed tube (Mallinckrodt, Inc., New York, USA). High frequency jet ventilation (HFJV) was commenced using a Penlon-Bromsgrove Jet Ventilator. Both the ventilator and entrainment circuit were supplied by a common blender (Bird Microblender, Bird Products Corp., Palm Springs, CA, USA) with a high pressure high flow outlet for the ventilator and a low pressure low flow outlet for the entrainment circuit which thus accurately control oxygen concentration (Fig. 2). Initially the ventilator was set to generate a rate of 120 breath.min⁻¹ at a driving pressure of 20 psi (137 kPa) and an inspiratory:expiratory ratio of 0.3. The fractional inspired oxygen concentration (F_{IO₂}) was kept at 1.0.

Anaesthesia was maintained with an infusion of propofol and an intravenous bolus of atracurium 20 mg. The propofol was started at a rate of 300 mg.h⁻¹ which was reduced to 200 mg.h⁻¹ after 30 min. After induction, epidural analgesia was provided by 5 ml bupivacaine (0.25%) followed by 30 µg fentanyl (in 3 ml saline). One further dose of atracurium 10 mg was given 1 h before the end of surgery.

ECG, arterial blood pressure, airway pressure, urinary output and oxygen saturation were monitored intra-opera-

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Fig 1. Pre-operative chest X ray showing lack of pulmonary markings in the right mid and upper zones.

tively. A left radial arterial line was inserted after induction and three arterial blood gas samples were taken at 10, 50 and 100 min after starting HFJV (Table 2). The F_{IO_2} was further reduced to 0.5 after the second blood gas estimation. Thereafter, the ventilator settings were kept unchanged until the end of the operation, even though a degree of hypercarbia was shown by the third blood gas estimation. Peak airway pressures did not exceed 9 cmH₂O throughout the procedure. Blood pressure and pulse rate remained stable throughout the operation and the oxygen saturation stayed at 99%.

At thoracotomy, a large apical communicating cyst was found. A right upper lobectomy had to be performed as the

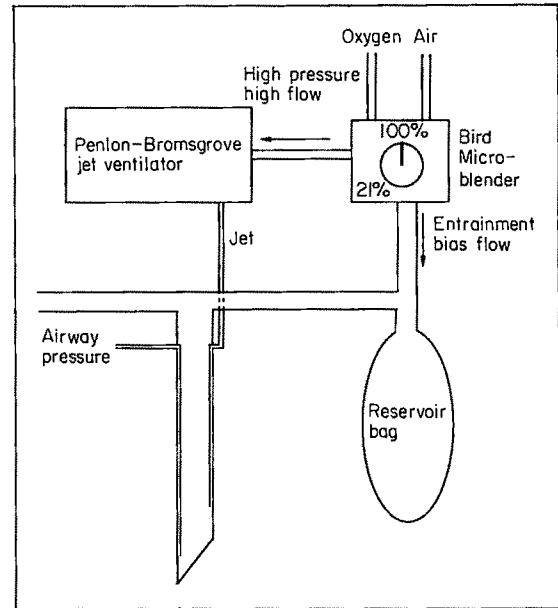


Fig. 2. The Bird Microblender can be adjusted to control oxygen concentration to the jet ventilator and the entrainment circuit simultaneously.

rest of the lobe was emphysematous. The anaesthetic technique provided excellent operating conditions, and blood loss was estimated to be 200 ml.

During closure of the chest, 5 ml of 0.125% bupivacaine with 25 µg of fentanyl was given through the epidural catheter. The propofol infusion was discontinued and the patient's trachea extubated 10 min later, when he was awake, breathing spontaneously and responding to commands. In the recovery room, he was pain-free and able to move all limbs. He was then transferred to the intensive care unit (ICU).

In the ICU, an epidural infusion of bupivacaine (0.125%) at 4 ml.h⁻¹ was commenced and good analgesia was achieved. This was increased to 5 ml.h⁻¹ the next day and continued until discharge to the ward.

In the immediate postoperative period, the patient

Table 1. Lung function tests.

	Predicted	Pre-operative	Postoperative	
			No broncho-dilator	With broncho-dilator
FEV ₁ ; l	2.18	0.5	0.86	1.05
FVC; l	2.85	1.45	1.6	1.7
PEF; l.min ⁻¹	421	90	160	165

FEV₁, forced expiratory volume in 1 s; FVC, forced vital capacity; PEF, peak expiratory flow rate.

Table 2. Arterial blood gas values.

			Intra-operative			Postoperative		
Pre-operative			10 min	50 min	100 min	Day 0	Day 3	Day 13
F_{IO_2}	air	nasal O ₂	1.0	0.8	0.5	0.3	0.24	air
pH	7.43	7.41	7.32	7.41	7.36	7.33	7.45	7.44
P_{aCO_2} (kPa)	4.8	5.2	8.07	6.49	6.83	7.55	4.0	6.2
P_{aO_2} (kPa)	8.0	11.4	63.7	37.5	10.5	15.34	9.2	8.5

became mildly hypercarbic (Table 2) and tachypnoeic with a respiratory rate of 22 breath.min⁻¹. He was using his accessory muscles of respiration and on auscultation air entry to the right lung was reduced. Two hours after extubation, he developed more severe respiratory distress which required re-intubation and intermittent mandatory ventilation. His trachea was extubated 3 h later and he remained stable on 30% oxygen by mask. Mask oxygen therapy was reduced to 24% on the third postoperative day, when he was discharged to the ward (Table 2). The patient's course in the ward was uneventful except for an air leak from the right chest, which closed spontaneously after 2 weeks. He was discharged home 3 weeks after his operation. The patient's pulmonary function tests before discharge showed a marked improvement over pre-operative values (Table 1), and his arterial blood gas values in room air were in the normal range for his age (Table 2).

Discussion

With improvements in the understanding and investigation of bullous lung disease, the number of patients with lung bullae who are likely to benefit from surgery will increase [5-8]. Resection of the giant bulla with the affected surrounding lung is the surgical procedure of choice, although simpler procedures such as thoracostomy drainage of the bulla may have a place [9,10]. Apart from the additional problems of underlying chronic lung disease, rupture of a bulla is a potentially life-threatening complication of general anaesthesia. The risks of expansion of a bulla or cyst (especially if non-communicating) when using nitrous oxide, and of barotrauma from positive pressure ventilation, are well known. Pavlin [1] recommends an anaesthetic technique which avoids nitrous oxide, allows the patient to breathe spontaneously, and uses a double-lumen tube to ventilate the non-bulla lung preferentially. Our present balanced technique of a propofol infusion, HFJV and epidural blockade, minimises the risk of barotrauma and also provides better operating conditions, gas exchange and analgesia.

Infusions of gamma-OH [11], Althesin [12] and ketamine [13] have been used to provide acceptable anaesthesia for patients with bullous lung disease, but each agent has its disadvantages and some are no longer available. Propofol has the advantage of a smooth induction, despite a few minor side effects such as pain on injection [14] and it may attenuate the haemodynamic response to intubation [15].

The use of HFJV has been reported in two patients with bullous lung disease who underwent surgery; one of bullectomy [13] and the other for coronary artery bypass surgery [12]. HFJV achieves much lower peak and mean airway pressures (and by extrapolation, alveolar pressure, as is evident clinically at thoracotomy) than conventional ventilation. Peak airway pressures in our patient did not exceed 9 cmH₂O. The minimal chest movement and relatively compliant lung achieved with HFJV provided a good surgical field. HFJV in our case was facilitated by the use of a Mallinckrodt 'Hi-Lo Jet' cuffed tube. This tube has two pilot ports; one at 5.5 cm from the tip which was used for jet ventilation and the other 0.5 cm from the tip which was used to measure airway pressure.

The use of a thoracic epidural is effective for analgesia during and after intrathoracic surgery [11]. Our patient

was haemodynamically stable intra-operatively and had a pain-free rapid recovery. The combination of bupivacaine and fentanyl in a continuous thoracic epidural infusion provides good quality postoperative analgesia [16]. Additional epidural opioids were not given in the ICU, as the cause of the sudden deterioration 2 h after surgery had not been established. A number of factors such as respiratory depression from epidural opioids, sputum retention and residual effects of anaesthesia may have been responsible. The patient was a high-risk case with very poor respiratory reserve.

In conclusion, a technique of balanced anaesthesia comprising total intravenous anaesthesia with propofol, HFJV with atracurium, and a thoracic epidural infusion, provided optimal peri-operative conditions for a high-risk patient who underwent surgical resection of a giant bulla. The risk of bulla rupture was minimised, while gas exchange was maintained. A rapid recovery with good postoperative analgesia were other advantages.

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CASE REPORT

Anaesthesia for intraperitoneal hyperthermic perfusion

G. SUDARSHAN AND D. CRAWFORD

Summary

Intraperitoneal hyperthermic perfusion is a method of regional chemotherapy shown to be effective in the prevention and treatment of peritoneal metastases. General anaesthesia for a patient who required this procedure is described. Guidelines for management are suggested, with particular emphasis on temperature control, fluid and electrolyte balance and postoperative care.

Key words

Cancer; chemotherapy, peritoneal metastases.
Anaesthesia; intraperitoneal hyperthermic perfusion.

Peritoneal metastases are a common problem associated with gastrointestinal cancer and no truly effective treatment is available. Hyperthermia is known to have antitumour effects and it also enhances the antitumour efficacy of certain cytotoxic agents. Whole body hyperthermia was used in the 1970s for treatment of sarcomas [1], but regional hyperthermia is better tolerated and there is now renewed interest in the effectiveness of hyperthermia in controlling the spread of abdominal tumours [2–5].

Intraperitoneal hyperthermic perfusion (IPHP) is a regional hyperthermic technique which has been used in some centres in the management of peritoneal cancer [3–6]. It involves closed peritoneal irrigation with a solution containing a cytotoxic agent. The perfusate (3–9 l) is instilled into the peritoneal cavity via a catheter inserted through the anterior abdominal wall. The temperature of the perfusate may vary from 42 to 52°C and the duration of the procedure is usually about 2–3 h.

The peritoneal cavity has a surface area of more than 2 m² and significant heat gain can occur during the procedure. Irrigation with such large volumes at high temperatures can impede diaphragmatic movement and lead to considerable discomfort. Hence general anaesthesia and controlled ventilation is required.

The anaesthetic implications of IPHP, however, have not been well documented therefore we report a patient with peritoneal metastases treated with IPHP under general anaesthesia.

Case history

A 65-year-old woman presented with evidence of peritoneal recurrence and ascites after undergoing radical resection of a gastric carcinoma 6 months previously. The diagnosis was confirmed by ultrasound and paracentesis. The ascites was drained and she was scheduled for IPHP under general anaesthesia. Her electrolytes were as follows: sodium 129 mmol.l⁻¹, chloride 101 mmol.l⁻¹, potassium 3.7 mmol.l⁻¹, urea 6.7 mmol.l⁻¹, creatinine 48 mmol.l⁻¹. She was premedicated with oral temazepam before arrival in the anaesthetic room.

After pre-oxygenation and application of cricoid pressure, anaesthesia was induced with thiopentone 200 mg and suxamethonium 100 mg. The trachea was intubated and intermittent positive pressure ventilation (IPPV) was instituted with 30% oxygen in nitrous oxide; ventilation was adjusted to maintain an end-tidal carbon dioxide concentration between 4.5 and 5%. Vecuronium was used to provide muscular paralysis and isoflurane was administered throughout the procedure. A radial artery cannula was inserted to facilitate blood sampling and to measure arterial pressure and a pulmonary artery (PA) catheter was inserted via the internal jugular route to measure the central pressures and the core temperature. The ECG, and nasopharyngeal and oesophageal temperatures were displayed continuously. The patient was placed on a thermostatically controlled electrical cooling mattress

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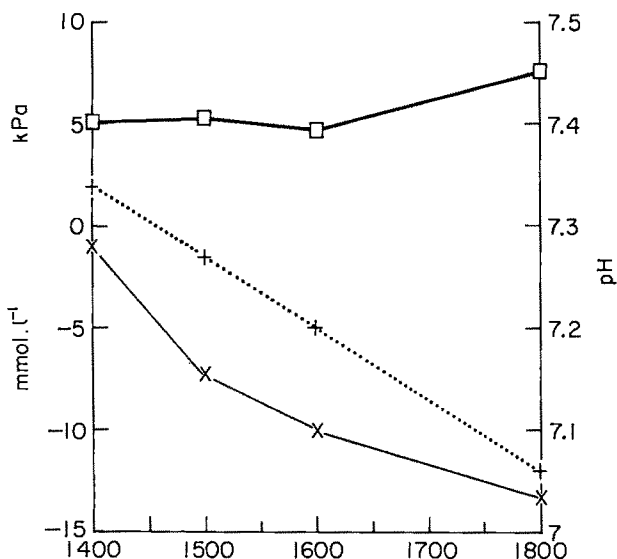


Fig 1. Acid base balance during intraperitoneal hyperthermic perfusion which started at 1500 h and ended at 1630 h. +, pH; $\square$ PCO_2 ; x, base excess.

(Hawksley) and ice packs were used to assist surface cooling.

The isoflurane concentration was increased and a small dose of sodium nitroprusside was given to produce peripheral vasodilatation and redistribution of heat from the core to the periphery, to induce a state of prehyperthermic hypothermia. Hyperthermic perfusion was started when the core temperature had fallen below 34°C. The peritoneum was initially primed with 2 l 0.9% saline and IPHP was performed with a closed irrigation using 3 l 0.9% saline containing mitomycin C 10 $\mu\text{g.ml}^{-1}$. Temperature measurements were made at the points of inflow and outflow on the abdominal wall. IPHP was performed for a period of 90 min; the temperatures at the inflow and outflow points were 47 and 40°C respectively. The combination of cooling mattress, ice packs and cutaneous vasodilatation was effective in maintaining the temperature in the pulmonary artery below 34°C. Filling pressures were maintained with 1 l 5% dextrose and 1 l Dextran 70 (6% in 5% dextrose) because of the possibility of significant sodium uptake from the perfusate.

During IPHP, periodic sampling of arterial blood gases was performed and a trend of metabolic acidosis became apparent through the procedure (Fig. 1). The nasopharyngeal temperature correlated well with the core temperature throughout the procedure, except during the rewarming phase. The oesophageal temperature, however, was easily influenced by the perfusate temperature and did not correlate with the measurements from other sites (Fig. 2). Rewarming was started about 30 min before the end of IPHP with the help of the warming mattress.

Neuromuscular blockade was reversed when the core temperature was 37°C. A peripheral nerve stimulator was used to confirm the adequacy of reversal and the patient was transferred to the recovery room after extubation. Half an hour later she became stuporous and peripherally cold. Blood gas analysis showed (F_{IO_2} 0.4): pH 7.06, P_{aCO_2} 7.6 kPa, P_{aO_2} 12.1 kPa, HCO_3^- 16 mmol.l^{-1} and a base excess (BE) of -10 mmol.l^{-1} . Her plasma sodium had decreased to 116 mmol.l^{-1} . Tracheal intubation was per-

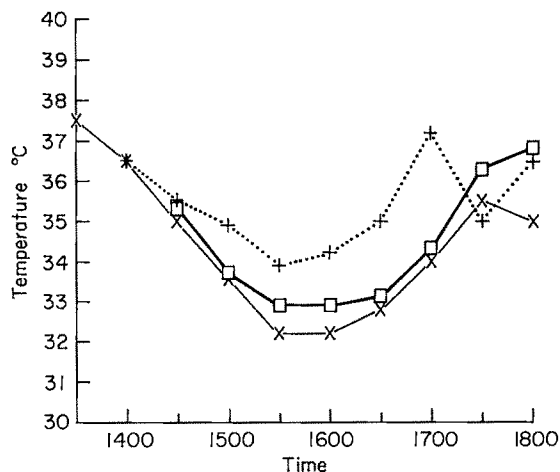


Fig. 2. Temperature trends in the oesophagus (+), nasopharynx (x), and pulmonary artery ($\square$) during intraperitoneal hyperthermic perfusion.

formed, IPPV was started and sodium bicarbonate 50 mmol was given. She was transferred to the intensive therapy unit (ITU), where her core temperature was found to be 34°C.

Sedation, IPPV and fluid restriction were maintained until the peripheral temperature was normal and the acidosis had resolved. Four hours later, when the plasma sodium was 124 mmol.l^{-1} , she was weaned off the ventilator without any problems. Apart from moderate abdominal discomfort and persistent ileus for 3 days, her subsequent stay was uneventful. The plasma sodium gradually increased to 129 mmol.l^{-1} over this period.

She was discharged from the surgical ward after 7 days, at which stage the only abnormality was a persistently low level of plasma albumin. She died 2 months later, with lymphangitis carcinomatosa, but without evidence of recurrence of her ascites.

Discussion

Intraperitoneal hyperthermic perfusion (IPHP) is gaining popularity in the western world as a method of prevention and treatment of peritoneal metastases following gastrointestinal cancer [7,8]. The procedure presents significant problems to the anaesthetist with regard to temperature control, fluid and electrolyte balance and postoperative care.

During IPHP there is a potential for heat gain from the peritoneal cavity. Several workers have reported a significant increase in pulmonary arterial (PA) temperatures (up to 40°C) despite pre-operative cooling [3,4]. The heat gained during the procedure depends upon the amount of heat supplied (i.e. the volume and temperature of the perfusate) and the surface area of the peritoneal cavity. Adhesions resulting from disease can impair both the heat transfer and the effectiveness of IPHP and some workers have used peritoneal expanders to promote an even distribution of the perfusate [6]. Accurate monitoring of temperature is essential.

The maximum core temperature achieved will be dependent on the pre-operative body temperature and this is the basis for induction of hypothermia before beginning IPHP. We elected to cool the patient to 34°C initially and to

monitor temperature at several sites. Measurements from the oesophageal site did not correlate with the core temperature measurements obtained from the thermistor placed in the pulmonary artery, presumably because the former was influenced by mixing of the perfusate in the peritoneal cavity. The nasopharyngeal temperatures more closely reflected PA temperatures throughout the procedure therefore the nasopharynx should be the principal site of temperature monitoring. Such monitoring should be continued well into the postoperative period.

Prolonged procedures which involve administration of substantial volumes of intravenous and intraperitoneal fluids to elderly patients require close monitoring. Measurement of central venous pressures are important for assessment of fluid balance and direct arterial pressure monitoring is necessary if sodium nitroprusside is used, and also provides access for regular blood gas sampling.

The absorption of sodium from the peritoneal irrigation was in fact negligible and the hyponatraemia in this patient was the result of administration of sodium-free fluids in a patient with mild hyponatraemia. We therefore recommend the use of sodium-containing fluids to maintain filling pressures throughout the procedure, with regular monitoring of plasma electrolytes and haematocrit. Metabolic acidosis (due to anaerobic glycolysis) is a recognised complication of mild to moderate hypothermia and is often self-correcting. The acidosis in this patient took nearly 4 h to resolve and required treatment with sodium bicarbonate. It is possible that tumour lysis may have been partially responsible for the severity of acidosis seen in this patient.

Postoperatively these frail patients are likely to have a labile body temperature, continuing acidosis, severe pain and ileus. We therefore recommend that these patients are transferred to an ITU for postoperative IPPV until body temperature is stable and acidosis has resolved.

In conclusion, anaesthesia for IPHP poses a significant challenge for the anaesthetist. We believe that we are still in the process of learning and more experience will be required before a reliable and safe anaesthetic technique for this procedure is established. There were disadvantages to the technique we used and it is possible that epidural analgesia could produce equally efficient vasodilatation in addition to providing excellent postoperative analgesia. We consider that temperature monitoring can be reliably achieved by the nasopharyngeal route. Central venous and

direct arterial pressure monitoring can be valuable aids in the management of fluid balance. These patients are prone to surgical complications (bowel perforation, side effects from cytotoxic drugs etc.) in addition to the ones outlined in this case report and will benefit from postoperative care in the intensive therapy unit for a period of 24–48 h.

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Fibreoptic intubation in cicatricial membranes of the pharynx

J. V. DIVATIA, S. M. UPADHYE AND R. SAREEN

Summary

Two patients presented with almost total obliteration of the pharynx. In one, a membrane developed after corrosive poisoning; in the other, the oropharynx was filled with a dense cicatrix in the sclerosing phase of rhinoscleroma. In both patients, a single opening in the membrane provided access to both the larynx and oesophagus. Fibreoptic intubation allowed both a thorough assessment of the pathology and subsequently the passage of a cuffed tracheal tube to secure the airway. To overcome the problem of respiratory obstruction while the fibrescope passed through the opening in the membrane, either rapid intubation, or a technique using pre-oxygenation and voluntary hyperventilation followed by breath-holding during bronchoscopy, was used. The thin calibre and manoeuvrability of the flexible fibreoptic bronchoscope makes fibreoptic intubation an excellent technique of airway management in cicatricial membranes of the pharynx.

Key words

Intubation, tracheal; fibreoptic.

Complications; hypopharyngeal stenosis, rhinoscleroma, caustic burns.

Tracheal intubation using the flexible fibreoptic bronchoscope (FFB) is a major advance in the management of difficult intubation [1]. Almost total obliteration of the oropharynx or hypopharynx by cicatricial membranes is an unusual occurrence but it may render conventional laryngoscopy and intubation impossible. Two such cases are presented; in both, fibreoptic intubation helped to overcome the problems in airway management.

Case histories

Patient 1

A 20-year-old, 54 kg male presented for evaluation of dysphagia after accidental ingestion of caustic soda 2 years previously. Barium swallow X rays showed delayed passage of barium through the hypopharynx and postcricoid region and diverticula in both piriform fossae. A fibreoptic oesophagogastroscope was introduced orally under topical anaesthesia. As it approached the 12 cm mark, the patient developed severe respiratory obstruction and cyanosis, which resolved as soon as the endoscope was withdrawn. Endoscopic examination revealed a cicatricial membrane extending across the pharynx at the orohypopharyngeal junction, with an elliptical hiatus measuring about 8 mm × 5 mm (Fig. 1(a) and (b)). This aperture provided sole access to the respiratory and alimentary tracts (Figs 2 and 3). It was decided to excise the membrane with a

carbon dioxide (CO₂) laser, enabling safe endoscopic examination whilst avoiding an otherwise inevitable tracheostomy. General anaesthesia was required. The pre-anaesthetic examination was unremarkable. Nutrition was well maintained through a feeding gastrostomy. There was no stridor at rest or on exertion.

An awake fibreoptic intubation was planned. After explaining the procedure to the patient, the airway was anaesthetised with 4 ml nebulised lignocaine 4%, started 30 min before the procedure. An intravenous infusion was set up and 0.6 mg atropine given intravenously. Electrocardiogram (ECG), heart rate, manual blood pressure, oxygen saturation (SpO₂) and end-tidal CO₂ (F_E/CO₂) were monitored. A 7 mm internal diameter (i.d.) red rubber tracheal tube was fitted over a 5.5 mm external diameter (e.d.) FFB. The FFB was introduced through the nose and the opening in the membrane was located. As it passed through the hiatus, respiratory obstruction developed. Within 20 s the FFB was manoeuvred into the trachea, the tracheal tube threaded over it and the FFB withdrawn. Anaesthesia was then induced with thiopentone and pancuronium and maintained with nitrous oxide, oxygen and halothane using controlled ventilation. After the cicatrix had been vaporized, neuromuscular blockade was reversed and the trachea extubated. Subsequently, a stricture of the middle third of the oesophagus was dilated under topical anaesthesia.

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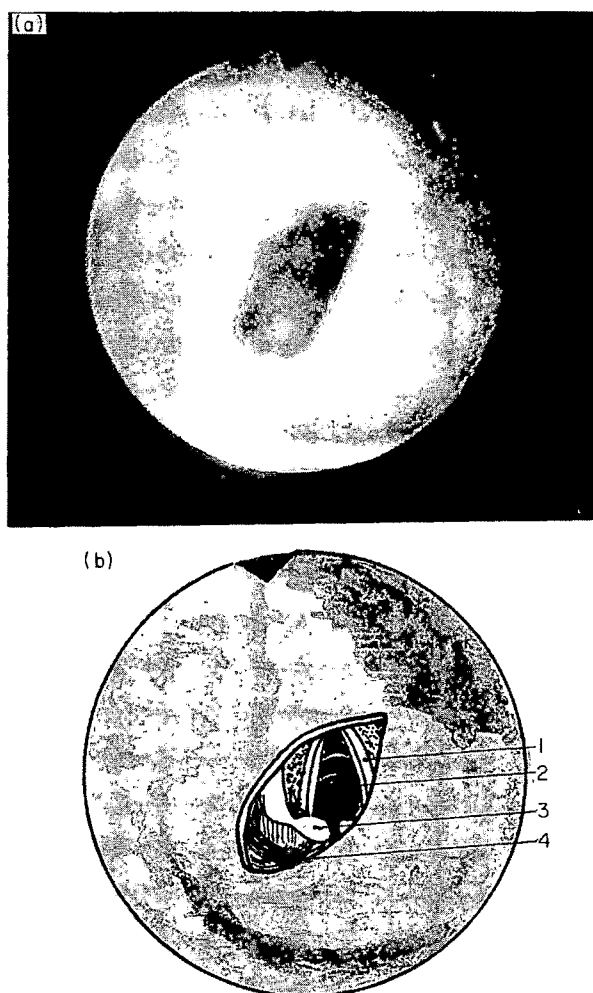


Fig. 1. (a) Endoscopic photograph of patient 1. The membrane is seen with its elliptical opening. The laryngeal structures are faintly visible. (b) Schematic diagram of (a). 1 = vocal cord, 2 = glottic aperture, 3 = arytenoid cartilage, 4 = piriform fossa.

Patient 2

A 56-year-old male patient, with a 17-year history of rhinoscleroma, presented with complaints of dysphagia, noisy breathing and dyspnoea on exertion. Pre-operative elective tracheostomy and excision of oropharyngeal adhesions had been performed on two occasions in the past.

On examination, mild inspiratory stridor was present. Both nares were obliterated by scar tissue. A cicatricial membrane filled the entire oropharynx, obliterating both anterior faucial pillars. A 4–5 mm opening was present at the junction of the membrane and the tongue (Fig. 4). There was another opening in the soft palate, but when the first aperture was occluded with a finger no air was felt to pass through the second. Laser excision of the cicatrix under general anaesthesia was planned.

One hour before the procedure the patient was premedicated with glycopyrronium 0.2 mg intramuscularly. Bilateral superior laryngeal nerve blocks were performed and 4% lignocaine 2 ml was injected intratracheally through the cricothyroid membrane. Diazepam 0.1 mg.kg^{-1} was given, so that the patient was sedated but arousable. Monitoring of ECG, heart rate, SpO_2 and

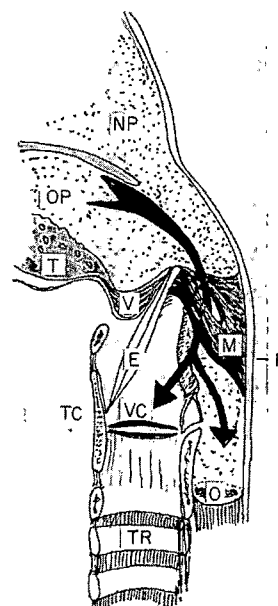


Fig. 2. Schematic diagram of a sagittal section through the nose, mouth, pharynx and larynx in patient 1. The membrane (M) extends from the vallecula (V) to the posterior pharyngeal wall (P) and involves the epiglottis (E). The arrows show that the opening is common to the larynx (VC = vocal cords) and oesophagus (O). NP = nasopharynx, OP = oropharynx, T = tongue, TC = thyroid cartilage, TR = trachea.

manual blood pressure was begun and an oral endoscopy airway inserted.

A snugly fitting 4.5 mm i.d. cuffed red rubber tube (e.d. 6.7 mm), covered with aluminium tape, was inserted through the oral airway into the communicating opening in the membrane. Oxygen 100% was delivered via a Magill system through the tube, while the patient was instructed to hyperventilate. A 3.5 mm e.d. FFB was introduced through the tube. The patient was asked to hold his breath in inspiration for 30 s while the passage below the opening was quickly assessed and found to be patent. The FFB was withdrawn, the patient was again asked to hyperventilate with 100% oxygen and the FFB re-inserted. At the third attempt the larynx and trachea were inspected and found to

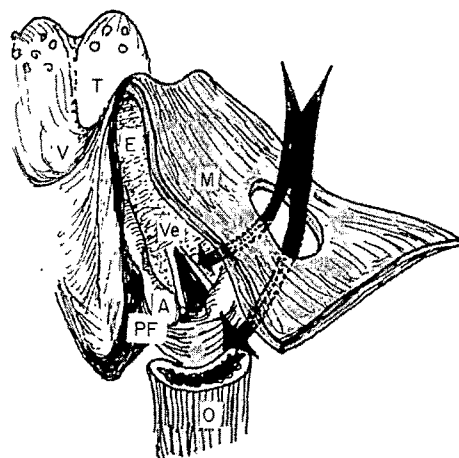


Fig. 3. Posterior view of the relations of the membrane in Figure 2. A = arytenoid cartilage, PF = piriform fossa. Ve = Vocal cord, M = membrane, O = oesophagus.



Fig. 4. Patient 2. Dense oropharyngeal cicatricial membrane in rhinoscleroma. The opening through which intubation was attempted is marked with an arrow. The larger opening above did not communicate with distal air passage.

be normal. The red rubber tube was passed over the FFB into the trachea. Anaesthesia was then induced with thiopentone and pancuronium and manual ventilation of the lungs was started. The oxygen saturation remained above 97% and $P_{a}CO_2$ below 5 kPa throughout the procedure, despite the periods of breath-holding and the small diameter of the tracheal tube.

After excision of the membrane, direct laryngoscopy could be performed easily. The 4.5 mm tube was removed and replaced with an 8 mm tracheal tube. The patient was discharged the next day, able to breathe and swallow comfortably.

Discussion

Stenosis of the pharynx secondary to scar tissue may occur after acute infections such as scarlet fever and gangrenous tonsillitis or after pharyngeal trauma or surgery, and chronic inflammations, notably syphilis and lupus vulgaris, may result in extensive scarring [2]. Hypopharyngeal stricture develops following ingestion of corrosive agents in 0.7% [3] to 6% [4] of cases and may be associated with oesophageal stenosis and partial or complete laryngo-tracheal stenosis [5].

Rhinoscleroma is a chronic infectious disease caused by *Klebsiella rhinoscleromatis*. In the cicatricial stage of the disease, dense fibrotic narrowing of the nose, pharynx, larynx or tracheobronchial tree takes place, and laryngeal and tracheal involvement is characterised by circumferential subglottic stenosis and obstruction by nodules and webs [6]. Involvement of the larynx, trachea and bronchi in rhinoscleroma has been reported in 36%, 20% and 7.5% of patients respectively [7].

The anaesthetic considerations in both patients presented above were similar, despite the diverse aetiologies. A small opening in the membrane provided the only approach to the respiratory and alimentary tracts. The size of the opening determined the degree of respiratory obstruction. Thus patient 2, who had a 4–5 mm opening, presented with stridor, whilst in patient 1, passage of an endoscope through the aperture precipitated severe respiratory obstruction. Attempts to pass dilators or even a nasogastric tube could have resulted in respiratory obstruction. An elective tracheostomy would have allowed endoscopy and

dilatation without respiratory obstruction and provided ideal conditions for anaesthesia, namely a secure airway, safety with the CO_2 laser and convenience for the surgeon. However, after consideration of the potential complications of tracheostomy and the fact that surgery would completely relieve airway obstruction, we were reluctant to perform a tracheostomy merely to simplify airway management, when alternative methods were available. In addition, the possibility of involvement of the trachea below the level of tracheostomy had not been excluded.

Blind intubation of the trachea was considered hazardous, because the patency of the pharynx distal to the membrane was not known and a tube introduced through the opening could not be expected to enter the trachea without manipulation or trauma. Retrograde intubation was not attempted since we felt that retrieval of the catheter through a narrow opening would be extremely difficult.

Transtacheal jet ventilation (TTJV) has been used as a method of ventilation during oral surgery [8] and in the management of difficult airway problems [9]. It has also been used to facilitate fiberoptic intubation under general anaesthesia [10]. However, our system for TTJV suffers from two major drawbacks. The jet injector is powered by unregulated central oxygen pressure from a wall outlet and there is no facility to monitor airway pressure during TTJV. In patient 2, had TTJV been used, obstruction to expiration by the dense membrane, by undiagnosed subglottic or tracheal membranes, or by blood and debris, was possible. The FFB was also expected to partially or totally occlude the orifice through which it was introduced. Severe barotrauma was likely under these conditions [11]. Therefore TTJV was not used.

We decided to perform awake fiberoptic intubation. In addition to the danger of actual or potential respiratory obstruction if the patient was asleep, active cooperation of the patients was required during intubation. Bronchoscopic assessment of the patency of the pharynx, larynx, trachea and bronchi was a fundamental step in airway management because of the propensity for cicatrization in both patients. It allowed us to proceed to tracheal intubation without fear of trauma or obstruction to the passage of the tube. The trachea was intubated with a cuffed red rubber tube covered with aluminium tape. This enabled us to maintain adequate oxygenation and ventilation, without risk of barotrauma and other minor complications of TTJV [9, 12]. The lungs were protected from inhalation of smoke and debris and there was little risk of tracheal tube ignition by the CO_2 laser [13].

The major hazard in both cases was respiratory obstruction as the FFB passed through the hiatus in the membrane. Although it could be rectified readily simply by withdrawing the FFB, continuous monitoring of arterial oxygenation and cardiac rhythm was of vital importance. In patient 1, although the degree of obstruction with the FFB was less than that with the oesophagogastroscope, rapid intubation of the trachea was crucial to the success of the technique. Use of a rigid bronchoscope may have averted this hazard in patient 1. Since the opening in the membrane was central, a rigid bronchoscope could have been passed through the opening to secure the airway and to ventilate the lungs, either with a jet injector or through a ventilating side-arm. In patient 2, however, rigid bronchoscopy was not possible. On the insertion of the FFB through the opening, the airway was totally obstructed.

Bronchoscopy was not likely to be rapid since the entire air passage had to be assessed before tracheal intubation. We therefore performed bronchoscopy while the patient held his breath for 30 s. We increased the breath-holding time by asking the patient to hyperventilate while breathing 100% oxygen before breath-holding [14], thus increasing patient safety and comfort.

Fibreoptic intubation has several limitations. It can only be used if the opening in the membrane admits a tracheal tube through which a FFB can be passed. It may be impossible if extensive scarring has occurred in the nose and oral cavity proximally, or beyond the membrane distally. In addition, fibreoptic intubation requires special skills and equipment which may not be universally available.

In summary, when the pharynx is occluded by a cicatricial membrane and a solitary opening provides access to the trachea, the size and position of the opening and the underlying pathology are both important considerations for anaesthesia. In cases in which assessment of the patency of the passage is essential or if the path to the trachea is not linear, the FFB is an invaluable aid. However, respiratory obstruction may occur during its passage. Transtracheal jet ventilation with monitoring of airway pressure, or ventilation through a rigid bronchoscope are other techniques which may be used. If the opening in the membrane is too small to permit such techniques, tracheostomy may be the final solution.

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CASE REPORT

Hypotension following valve replacement surgery in carcinoid heart disease

S. K. HAMID AND D. N. F. HARRIS

Summary

We report the use of adrenaline in a 62-year-old woman with carcinoid heart disease who underwent double valve replacement. She was given an intravenous infusion of octreotide throughout the peri-operative period. Following the termination of cardiopulmonary bypass she developed profound hypotension which proved to be refractory to various therapies but responded to adrenaline. Adrenaline is said to be contraindicated in carcinoid syndrome, but we would suggest the consideration of its use in such cases where the hypotension may result from myocardial depression rather than from a carcinoid crisis.

Key words

Complications; hypotension, carcinoid heart disease. Sympathetic nervous system; adrenaline.

Carcinoid tumours arising from enterochromaffin cells are fairly common. The carcinoid syndrome, however, characterised by flushing, bronchospasm, diarrhoea and right sided heart lesions, is relatively rare. It is usually seen in cases in which there are hepatic metastases, when the tumour drains directly into the systemic circulation, and in carcinoid tumours of the lung. The tumours release a variety of substances which can include 5-hydroxytryptamine (5HT), bradykinins, histamine, prostaglandins and neuropeptides, such as substance P and neurokinin A.

Right heart failure in patients with carcinoid heart disease signifies a poor prognosis. There have been reports in the literature of eight patients undergoing double valve replacement; two of these patients died in the postoperative period, one at 5 days following operation from multi-organ failure and one at 1 month from bronchopneumonia [1].

Problems with haemodynamic instability and bronchospasm can make surgery and anaesthesia difficult [2]. Previous case reports stress the importance of preparing patients adequately in the pre-operative period [3, 4]. This includes maintaining the patient on drug therapy to block either the release of vasoactive substances from the tumour or the effects of the vasoactive substances themselves [5, 6].

We report the anaesthetic management of a patient with carcinoid heart disease and right heart failure undergoing

double valve replacement, in whom we had to use adrenaline following cardiopulmonary bypass (CPB).

Case history

A 62-year-old woman was seen initially in December 1989. She presented with abdominal pain, weight loss, fatigue and diarrhoea. On clinical examination her liver was noted to be enlarged and computerised tomography (CT) showed that it contained widespread metastases. Liver biopsy revealed the tumour to be of neuroendocrine origin but numerous investigations failed to locate the primary tumour. She was commenced on cyproheptadine and ketanserin therapy but was unable to tolerate the cyproheptadine because it made her drowsy, therefore she was started on a somatostatin analogue (octreotide).

In November 1990 hepatic embolisation was performed, which resulted in some relief of symptoms. In June 1991 she started to develop signs and symptoms of right heart failure. Echocardiography showed the tricuspid and pulmonary valves to be thickened with reduced excursions. Right heart catheter studies showed severe tricuspid and pulmonary valve regurgitation therefore it was decided to replace both valves.

Serum urea and electrolytes and liver function tests were

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within normal limits except for a slightly increased bilirubin ($22 \mu\text{mol.l}^{-1}$) and alkaline phosphatase (169 iu.l^{-1}). Urinary 5-hydroxyindoleacetic acid (5HIAA) excretion was 603 mg.24h^{-1} . Electrocardiogram (ECG) showed sinus rhythm with frequent atrial ectopics. The chest X ray showed the heart size to be at the upper limit of normal but was otherwise unremarkable.

The patient was given cyproheptadine 4 mg three times a day, which had been restarted on this hospital admission, and octreotide $100 \mu\text{g}$ three times a day subcutaneously. Since she had previously been receiving morphine for analgesia, the patient was premedicated with lorazepam 2 mg orally, morphine 10 mg and droperidol 2.5 mg intramuscularly, 2 h before surgery. Oxygen, 4 l.min^{-1} via face-mask, was also given at this time.

Routine monitoring of intra-arterial pressure, 5 lead ECG and pulse oximetry was begun. Following induction of anaesthesia the right internal jugular vein was cannulated for central venous pressure monitoring and the CVP was 15 mmHg.

Intravenous infusions of octreotide at $100 \mu\text{g.h}^{-1}$ and aprotinin at $100\,000 \text{ u.h}^{-1}$ were started and maintained throughout anaesthesia, in order to suppress release of mediators and to antagonise their effects. Vancomycin 1 g, flucloxacillin 1 g and cefuroxime 1.5 g were administered.

Anaesthesia was induced with midazolam 4 mg and fentanyl 1 mg. Neuromuscular blockade was achieved with pancuronium 8 mg and intermittent positive pressure ventilation (IPPV) instituted to maintain an end-tidal CO_2 of 4.5–6.0 kPa. Anaesthesia was maintained with isoflurane (0.2–0.8%) in 50% oxygen in nitrous oxide and increments of fentanyl to a total dose of 1 mg.

Induction of anaesthesia was uneventful. Manipulation of the heart prior to cannulation led to profound hypotension (systolic BP < 50 mmHg) on two occasions before the institution of cardiopulmonary bypass (CPB). This resolved spontaneously when the surgical manipulation was stopped.

The patient was cooled to 30°C . Both the tricuspid and pulmonary valves were found to be thickened. The tricuspid valve was replaced with a porcine valve and the pulmonary valve with an aortic homograft. The bypass time was 180 min.

Following termination of CPB there was profound hypotension in the absence of flushing, which suggested that it may not have been the result of a carcinoid crisis. The CVP was 15 mmHg, the heart could be seen to be adequately filled and the pulmonary artery was tense on palpation. The patient's nasopharyngeal temperature was recorded as 37.5°C , there was no acidosis and the serum potassium concentration was 4.9 mmol.l^{-1} . The hypotension did not respond to several boluses of methoxamine (12 mg in total) or calcium chloride 10% 5 ml. A bolus of adrenaline ($5 \mu\text{g}$) was cautiously given. This temporarily improved the arterial pressure without causing flushing or signs of vasodilatation. A further bolus of adrenaline was given and this again resulted in improvement of the arterial pressure. An infusion of adrenaline was started and the rate was titrated against the haemodynamic response. Postoperatively the patient received IPPV in the cardiac intensive care unit and the adrenaline was progressively reduced and stopped after an hour. Her postoperative period was complicated by three episodes of flushing and profound hypotension overnight, 8 h after the adrenaline had been stopped, but these

resolved spontaneously. She was continued on reducing doses of octreotide intravenously and cyproheptadine via her nasogastric tube.

Discussion

Carcinoid heart disease develops more slowly than the other manifestations of the syndrome. It involves fibrosis of the endocardium, especially of the right side of the heart. Right ventricular failure is one of the major causes of death in patients with carcinoid syndrome. Valve replacement can improve the haemodynamics in these patients [7] but its value in prolonging life has not been proven [1]. Furthermore, there have been two case reports of bioprosthetic valves subsequently being affected by the carcinoid process [8, 9].

Most reports of the anaesthetic management of carcinoid syndrome stress the importance of treatment with drugs which block the vasoactive substances released by the tumour, and this patient was continued on the 5HT antagonist cyproheptadine. Octreotide, a somatostatin analogue which blocks hormonal release and inhibits the action of circulating peptides, was also used. It is now the mainstay of medical treatment [10]. Whilst this produces cardiovascular stability during normal activity it does not guarantee stability during anaesthesia and surgery. Morphine is said to be relatively contraindicated in carcinoid syndrome because of its potential for histamine release but it was used as premedication in our patient because she had received it before without adverse effects.

Catecholamines are also said to be contraindicated in carcinoid syndrome since they can cause release of tumour kallikrein which activates bradykinins, resulting in hypotension. They can also cause the release of 5HT which potentiates the vasoconstrictor responses of blood vessels to catecholamines. Methoxamine and angiotensin have been recommended as the drugs to treat the hypotension which results from vasoactive peptides in carcinoid.

Marsh and colleagues reported the use of adrenaline in carcinoid crisis during anaesthesia for a laparotomy [11]. This was unsuccessful in treating the crisis, which only responded to the administration of a somatostatin analogue.

Hypotension after CFB, however, may result either from the carcinoid syndrome or from myocardial depression subsequent to the cardiac operation itself. If hypotension is unresponsive to standard treatment with optimal volume replacement, correction of electrolyte abnormalities and methoxamine, more powerful agents must be considered. Angiotensin has been suggested, but it does not possess an inotropic action like adrenaline, which may be a more suitable drug for primary myocardial depression. It has been used successfully before at this hospital. There was no flushing associated with our patient's hypotension, a cautious trial of adrenaline produced an immediate beneficial response, and subsequently an adrenaline infusion resulted in a sustained improvement. We therefore suggest that the use of adrenaline should be considered in such cases, provided that direct arterial pressure is monitored and a small initial bolus of the drug is given to assess the patient's response. If the cause of the hypotension is thought to be release of vasoactive substances and flushing is observed, the hypotension is frequently transient and recovers spontaneously. In this situation the use of adrenaline would not be appropriate. Aprotinin, a kallikrein

trypsin inhibitor, also helped reduce blood loss, which was small in this operation. Although previous reports have not mentioned problems of excessive bleeding, aprotinin would appear to be an ideal agent for use in cardiac surgery in carcinoid syndrome because of its dual role.

In conclusion, hypotension in patients with carcinoid syndrome may not always be caused by a carcinoid crisis. We report the rational and successful use of adrenaline in a 62-year-old woman with carcinoid heart disease who had refractory hypotension following cardiopulmonary bypass.

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CASE REPORT

Caudal blockade in the management of aortic thrombosis following umbilical artery catheterisation

D. M. HARGREAVES, P. M. SPARGO AND R. A. WHEELER

Summary

A neonate, with a postconceptual age of 29 weeks, suffered thrombosis of the aorta as a consequence of umbilical artery catheterisation. This resulted in ischaemic lesions of the lower limbs and buttocks. Part of the management consisted of the insertion of an extradural catheter, via the caudal route, which provided good pain relief and may have improved lower limb blood flow.

Key words

*Anaesthetic techniques, regional; caudal.
Arteries; umbilical artery catheterisation.
Complications; aortic thrombosis.*

Umbilical artery catheterisation is a commonly used procedure in pre-term neonates to facilitate monitoring and blood sampling. Complications include aortic thrombosis, which carries a high mortality [1, 2]. Standard management of aortic thrombosis may include surgical thrombectomy, thrombolytic therapy and control of cardiac failure. We report the use of continuous caudal blockade in the management of a neonate of 29 weeks post-conceptual age, who presented with this complication.

Case history

A 1.3 kg girl was delivered by emergency Caesarean section at 29 weeks gestation. She was the heaviest of triplets, whose mother had been admitted that day for bedrest following the onset of hypertension and proteinuria. Premature labour had started the same day. She cried at delivery, but rapidly became apnoeic with a decreasing heart rate. Intubation and intermittent positive pressure ventilation (IPPV) through a size 2.5 tracheal tube were performed; she was then transferred to the special care baby unit (SCBU) for further management.

To facilitate monitoring and treatment, an umbilical artery catheter was inserted using a standard technique, at 2 h of age. The catheter tip was confirmed by X ray to be at the level of T₁₂. Three hours later the nursing staff noticed a

blue discoloration which extended from below the umbilicus to both legs. Pedal pulses were diminished and the legs felt cool. A diagnosis of aortic thrombosis was made and the catheter was removed. Doppler studies showed flow to the bifurcation of the aorta, but none below the femoral arteries on either side. Ultrasound scan of the head revealed bilateral Grade 1 intraventricular haemorrhages. At this stage, the treatment options included anticoagulant therapy with either heparin or streptokinase or surgical thrombectomy. In view of the potential for worsening the intraventricular haemorrhages, these options were considered to be contraindicated and conservative management, which consisted of IPPV, analgesia with a morphine infusion, intravenous fluids and antibiotics, was initiated.

During the next 3 days there was a deterioration in the ischaemic appearance of the buttocks and legs. The patient was haemodynamically stable, but there was an increase in the serum urea and creatinine concentrations (10.8 mmol.l⁻¹ and 213 µmol.l⁻¹ respectively). Respiratory function improved. At 5 days it was decided to transfer her to the regional paediatric surgical centre.

On arrival, she was breathing spontaneously on air. Both legs were generally mottled in appearance with several frankly ischaemic areas (Fig. 1). There were also ischaemic lesions on the perineum and buttocks. She showed minimal spontaneous movements of her legs, but despite receiving a

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Fig. 1. Initial appearance showing diffusely mottled lower limbs and discrete ischaemic lesions more marked on the right.

morphine infusion she was obviously distressed when her legs were handled, with crying, tachycardia and tachypnoea. The anaesthetic department were consulted about her analgesia and the use of extradural analgesia was considered to be of possible benefit. A 25-gauge needle (B-D Microlance) was inserted under aseptic conditions through the sacral hiatus into the extradural space with the patient in the left lateral position and 1.5 ml of a 0.17% plain bupivacaine solution (2 ml 0.25% bupivacaine with 1 ml saline) was injected through a bacterial filter. The effect of this procedure was to reduce the pulse rate from 210 to 160 $\text{beat}\cdot\text{min}^{-1}$, the respiratory rate from 90 to 60 $\text{breath}\cdot\text{min}^{-1}$ and to allow handling of the legs with appreciably less distress. In addition, it was noted that there was an improvement in the discoloration of the legs. These benefits lasted for 6 h.

An extradural catheter was inserted via the caudal route for further analgesia to avoid the need for repeated skin puncture. A 20-gauge Critikon cannula was used to insert a Portex paediatric extradural catheter (external diameter 0.63 mm) through the sacrococcygeal membrane and 5 cm into the extradural space. A second, similar dose of bupivacaine was given through the catheter with further beneficial results. An extradural infusion was started at the rate of 0.1 $\text{ml}\cdot\text{h}^{-1}$ of 0.25% bupivacaine, which was then increased to 0.2 $\text{ml}\cdot\text{h}^{-1}$ to produce the desired analgesic effect. In view of the proximity of the sacral hiatus to the perineum, we considered it unwise to leave the catheter in place for more than 48 h: following catheter removal the tip was sent for bacterial culture, which proved to be negative.

A bone scan suggested infarction of her femoral epiphyses and there continued to be no spontaneous movements of her legs. Spinal cord infarction was suspected. The ischaemic lesions on her buttocks and legs showed signs of resolution, but the area over her right fifth digit became demarcated by gangrene. This digit was subsequently amputated.

Eight weeks later she had started to show spontaneous movements of her legs but she still had a neurogenic bladder which required intermittent catheterisation.

Discussion

To our knowledge, the use of caudal analgesia in the management of neonatal aortic thrombosis has not previously been reported, nor has it been described in a neonate with such a low post-conceptual age. Aortic

thrombosis following umbilical artery catheterisation is rare with an incidence of less than 1% [1]. Unfortunately, it often has catastrophic consequences [2, 3]. Some authors consider that, without surgical thrombectomy, the condition is almost invariably fatal [1, 4]. Death is usually secondary to cardiac and renal failure. Thrombectomy was lifesaving in six out of seven neonates [1]. More recent work reports successful nonsurgical management in three cases [5]. This has involved the use of high dose antihypertensive medication, including hydralazine, propranolol, methyldopa and sodium nitroprusside, in addition to diuretics. In patients who survive there is a high incidence of renovascular hypertension and discrepancies in leg growth [6].

There is little doubt that the premature neonate feels pain, responds to painful stimuli and exhibits a stress response [7]. This stress response involves release of catecholamines, cortisol and antidiuretic hormone, which results in a tachycardia, vasoconstriction, increased metabolic rate and oxygen demand, salt and water retention and hyperglycaemia. It has been shown that regional anaesthesia, by both the subarachnoid and extradural route, delays and reduces the stress response to surgery, by blocking afferent impulses [8].

The potential benefits in this case include a direct vasodilator effect on peripheral vessels as a result of sympathetic blockade and an indirect effect secondary to decreased catecholamine release. In addition, the analgesia provided has less risk of respiratory depression than that associated with the use of opioid analgesic drugs.

Caudal blockade, in association with light general anaesthesia, is practised widely in patients above the age of 6 months, especially for operations below the level of the umbilicus. Excellent postoperative analgesia is provided and there is more rapid recovery from the effects of general anaesthetic agents. The procedure is reliable and safe, with a low incidence of complications [9]. There are few reports of its use in the neonatal period, especially in the premature neonate. The technique has been described for the repair of inguinal hernia in a premature, ventilator-dependent neonate of 32 weeks gestation [10] and in a series of infants with a post-conceptual age of less than 53 weeks [11].

The technique of continuous caudal anaesthesia is simple and easy to perform, but intravenous access and full resuscitative equipment are essential. A strict aseptic routine should be followed, the catheter should be passed gently into the epidural space and aspirated for signs of cerebrospinal fluid. A bacterial filter should be attached to the end of the catheter before injection of local anaesthetic. The specific problems associated with the caudal approach in this age group are the risk of inadvertent venepuncture, because the sacral venous plexus is closer to the point of entry of the needle than in older patients and the possibility of subarachnoid puncture, since the dural sac reaches a lower level in the newborn [12]. There is a risk of bacterial contamination because of the proximity of the perineum; for this reason it would seem prudent to remove the catheter as soon as possible [13].

Infants less than 3 months old have decreased liver blood flow and immature enzyme systems, which result in slower metabolism of amide local anaesthetics compared with older age groups. Lower plasma levels of serum albumin and α_1 -acid glycoprotein reduce drug binding and further increase the risk of toxicity [12]. However, a larger

volume of distribution for local anaesthetics in the neonate may confer some protection.

Sympathetic blockade of the lower limb has a variable and unpredictable effect in aorto-iliac occlusive disease in the adult; there is evidence that it is of benefit in patients with trophic changes and helps to protect from gangrene [14].

Although it is impossible to conclude that the use of caudal epidural blockade was beneficial to the specific underlying problem in this case, it undoubtedly helped in the overall management. Its use should be considered in this condition, especially if more standard treatment is contraindicated.

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CASE REPORT

Thoracic epidural anaesthesia and primary pulmonary hypertension

P. ARMSTRONG

Summary

Patients with primary pulmonary hypertension occasionally present for surgery. Anaesthesia requires continuous cardiovascular monitoring and maintenance of stable pulmonary and systemic haemodynamics. The management of a patient with severe pulmonary hypertension, undergoing open lung biopsy under a combination of general anaesthesia and thoracic epidural analgesia is reported and the problems of primary pulmonary hypertension are discussed.

Key words

*Complications; primary pulmonary hypertension.
Anaesthetic techniques; epidural thoracic.*

Individuals with primary pulmonary hypertension, a rare disease of the pulmonary vasculature, occasionally present for anaesthesia. One of the principal problems is to prevent increases in pulmonary arterial pressure (PAP), which result in right ventricular strain. We describe a patient in whom thoracic epidural analgesia provided suitable operative and postoperative conditions.

Case history

A 31-year-old black female was admitted to hospital for a diagnostic open lung biopsy. She had no significant past medical history, was a nonsmoker and had no known allergies. During the preceding 9 months she became easily fatigued, and had noticed decreased tolerance to exercise such that she was able to walk only a few yards on the flat. More recently she complained of breathlessness at rest, but in the absence of chest pain or haemoptysis.

On examination she was found to be overweight (95 kg), had a tachycardia of 110 beat.min⁻¹ and was dyspnoeic at rest. No other abnormalities were found apart from a loud pulmonary second sound on auscultation. Full biochemical, haematological and coagulation tests were normal. Electrocardiography (ECG) showed a sinus tachycardia with evidence of right ventricular hypertrophy and right axis deviation. Chest X ray revealed enlarged pulmonary arteries. Arterial blood gases whilst breathing room air were: pH 7.52, P_{aCO_2} 4.1 kPa, P_{aO_2} 7.4 kPa, bicarbonate 22 mmol.l⁻¹ and SO_2 of 88%. Lung function tests showed a vital capacity of 5.3 l and a FEV₁/FVC of 87%. Echocardiography demonstrated severe right ven-

tricular hypertrophy and a normal ejection fraction. Pulmonary catheterisation showed a PAP which exceeded systemic arterial pressure (Table 1), and did not respond to pulmonary nitroglycerin. Angiography showed severe proximal narrowing of the pulmonary arterioles. The patient was taking oral nifedipine, with little beneficial effect.

Premedication was with lorazepam 2 mg orally. On arrival in the anaesthetic room her arterial saturation was 86%. A 20-G left radial arterial cannula and a right internal jugular pulmonary catheter were inserted and haemodynamic measurement demonstrated an increase in the already high pulmonary–systemic (P–S) pressure difference. A thoracic epidural catheter was inserted in the midline at the T₅/T₆ interspace, using loss of resistance to saline, and a test dose of 3 ml 0.5% bupivacaine plain was given. After 5 min, a further 4 ml of 0.25% bupivacaine and 100 µg fentanyl were injected and this resulted in a slight improvement in the P–S pressure differences.

Induction of anaesthesia was accomplished with fentanyl 100 µg, thiopentone 200 mg, atracurium 30 mg, topical lignocaine spray to the vocal cords and insertion of a Portex 8.0 mm cuffed tracheal tube. Anaesthesia was maintained with 50% oxygen in air and isoflurane (1–2%) and ventilation was controlled to produce an end-tidal PCO_2 of 4.5 kPa. A further bolus of fentanyl 50 µg and atracurium 15 mg was given after 30 min. Surgery was completed in 80 min and the tracheal tube was removed when the patient was awake. Postoperatively, pain relief was given using an epidural infusion of 5 ml 0.125% bupivacaine and 20 µg fentanyl hourly. Pulmonary arterial pressures immediately

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Table 1. Pulmonary and systemic haemodynamic data.

	Before surgery	Before epidural	Before induction	After induction	Maintenance (min)			After extubation	Recovery (h)	
					20	40	60		4	12
PAP; mmHg	153/76	166/85	146/70	144/72	148/78	153/84	149/73	158/80	161/82	153/74
Mean PAP; mmHg	102	110	95	92	93	97	92	104	96	100
BP; mmHg	127/67	122/70	119/65	117/71	121/80	125/78	128/77	136/85	138/80	132/77
Mean arterial pressure; mmHg	88	85	80	77	91	95	93	101	97	85
CVP; mmHg	26	28	22	24	25	23	26	28	22	23
PAWP; mmHg	57	64	55	51	48	48	53	57	51	63
SVR; dyn.s.cm ⁻⁵	—	894	987	1034	1147	1175	1050	1123	1395	1078
PVR; dyn.s.cm ⁻⁵	—	721	680	800	782	800	611	723	874	643
QT; l.min ⁻¹	—	5.1	4.7	4.1	4.6	4.9	5.1	5.2	4.3	4.6
Sao ₂ ; %	88	86	87	98	99	99	97	92	93	91
Pe'CO ₂ ; kPa	—	—	—	4.6	4.4	4.3	4.6	—	—	—

PAP, pulmonary artery pressure; BP, systemic blood pressure; CVP, central venous pressure; PAWP, pulmonary artery wedge pressure; SVR, systemic vascular resistance; PVR, pulmonary vascular resistance; QT, cardiac output; Sao₂, oxygen saturation; Pe'CO₂, end-tidal carbon dioxide.

postextubation, and at 4 and 12 h are as shown in Table 1. The infusion provided sufficient pain relief that no other drugs were required. The diagnosis of primary pulmonary hypertension with irreversible plexogenic arteriopathy was made from the biopsy.

Discussion

Pulmonary hypertension exists when the systolic and mean pulmonary arterial pressures exceed 30 and 20 mmHg respectively. There are many causes of pulmonary hypertension (Table 2) and the diagnosis of primary pulmonary hypertension is made when other causes have been excluded. In pathophysiological terms, pulmonary artery pressure is dependent on three factors; pulmonary blood flow, left atrial pressure and pulmonary vascular resistance (PVR). Pulmonary hypertension can result from an abnormal increase in any of these. Two main pathological types are found; postcapillary obstruction (pulmonary venous hypertension) and precapillary obstruction (pulmonary arterial hypertension), which result from

increases in left atrial pressure and pulmonary blood flow respectively. In contrast, in primary pulmonary hypertension, a plexogenic pulmonary arteriopathy is often seen, although this is often accompanied by evidence of thrombo-embolic disorders [1]. The main changes occur in the smooth muscle layer of the pulmonary arterioles, which results in initial functional (reversible) vasoconstriction, but which progresses to muscle hypertrophy and degenerative changes which are irreversible.

The true incidence of primary pulmonary hypertension is unknown, but it occurs in 1% of autopsies on patients in whom cor pulmonale had been diagnosed. In young adults, it is primarily a disease of females, with a sex ratio of 4:1 [1], but in older age groups, the sex incidence is similar [2]. It is sometimes familial [3] and associations have been reported with Raynaud's phenomenon [4] and liver cirrhosis [5].

Primary pulmonary hypertension is of clinical importance because it predisposes to the development of right ventricular overload and failure (cor pulmonale). Atherosclerosis occurs and intrapulmonary shunts appear. Clinical manifestations of primary pulmonary hypertension are identical to those of pulmonary hypertension secondary to other causes. Mild and moderate degrees of primary pulmonary hypertension are usually asymptomatic although the disease can progress rapidly to the stage of cardiorespiratory failure. The patient complains of severe fatigue, dyspnoea, haemoptysis and chest pain [6]. Examination may show evidence of a low output state, accentuation of the pulmonary component of the second heart sound, right ventricular hypertrophy and failure. The onset of atrial fibrillation tends to be a late event. Early X ray changes include enlargement of the pulmonary arteries, followed by right ventricular hypertrophy. Electrocardiographic changes show signs of right ventricular and right atrial hypertrophy. Echocardiography is of little help in the detection of mild to moderate disease [7] and in these cases lung scan is usually normal [6]. Cardiac catheterisation combined with pulmonary angiography produces the best information on the degree of pulmonary hypertension, the cardiac reserve and the effects of pulmonary vasodilator therapy. In primary pulmonary hypertension, the right ventricular and pulmonary artery pressures are greatly increased, often to above systemic pressures, and in the

Table 2. Causes of pulmonary hypertension.

1. Pulmonary venous hypertension
(a) Left atrial outflow obstruction
e.g. mitral and aortic valve disease
pericarditis
left ventricular failure
(b) Pulmonary venous occlusive disease
e.g. pulmonary venous thrombosis
2. Pulmonary arterial hypertension
(a) Hyperdynamic circulation
e.g. left to right shunts, chronic anaemia and hypoxia, liver failure
(b) Vasoconstrictive causes
e.g. primary pulmonary hypertension, Eisenmenger syndrome
(c) Viscous cause
e.g. polycythaemia
(d) Obstructive causes
e.g. recurrent emboli, sickle cell disease, chronic obstructive airways disease, pulmonary fibrosis
(e) Reactive vascular disease
e.g. sarcoidosis, Goodpasture's syndrome, polyarteritis nodosa

presence of right ventricular failure, there are increased end-diastolic and atrial pressures. The wedge pressure is falsely high as a result of pulmonary capillary obstruction, and left atrial pressure is low to normal. Occasionally, lung biopsy is necessary for diagnosis and open biopsy is the preferred method since the samples obtained by transbronchial biopsy are usually inadequate.

The clinical course follows an inexorable decline, with most patients developing right heart failure and death within 5 years of diagnosis, although cases have been documented of survival up to 20 years. Inadequate pulmonary and right ventricular function and reduced arterial oxygen saturation levels signify a poor prognosis [1]. Once vascular changes supervene, local complications rapidly increase the severity of the disease. This is especially true for thrombotic occlusion, which occurs in 30–50% of cases [8].

Medical treatment of primary pulmonary hypertension is aimed at reducing both PVR and PAP. Pulmonary vasodilator therapy may help in patients with reversible vasoconstriction. However, two major problems may occur with their use; a secondary reduction in systemic vascular resistance (SVR) with systemic hypotension, and reversal of the hypoxic pulmonary vasoconstriction resulting in increasing hypoxia. Drugs which have been used include hydralazine, tolazoline, isoprenaline, diazoxide and phentolamine [9]. Intravenous nitroglycerin is effective in some patients [10], as are prostaglandin_{E₁} [11] and prostacyclin [12]. However, the overall response to vasodilators is poor and there is little evidence that they improve mortality [2].

Patients with primary pulmonary hypertension may present for anaesthesia for a variety of reasons; coincidental emergency or elective surgery, diagnostic procedures and pregnancy. Initial assessment of the severity of the disease can be made from a history and clinical examination. Cardiological investigations provide pressure measurements and an assessment of the degree of reversibility. The principal requirements during anaesthesia are to prevent increases in pulmonary hypertension and to avoid major haemodynamic changes, since the combination of increases in PVR and secondary right ventricular failure may be fatal [13]. The pregnant patient with primary pulmonary hypertension is at particularly high risk, with a mortality rate of 50% [14].

Full invasive cardiovascular monitoring, including continuous measurements of both pulmonary and systemic arterial pressures, is required.

The disease is affected both by the physiological changes which occur during anaesthesia and the anaesthetic drugs themselves. In addition, the pulmonary and systemic vasculature can react differently to the same stimulus, probably in order to match the ventilation/perfusion needs of the lung. A decrease in P_{aO_2} below 13.3 kPa results in an increase in PVR, which reaches a maximum when the P_{aO_2} has fallen to 4 kPa [15]. Similar undesirable changes in PVR occur with hypercarbia [16], acidosis [17], high airway pressures [18] and positive end-expiratory pressures [19]. Stress and pain increase PVR as a result of centrally mediated sympathetic effects and secondary to the release of circulating catecholamines. Tracheal intubation increases PAP and causes right ventricular strain [20].

Inhalational agents are negative inotropes and decrease PVR. In normal individuals, PAP is not affected by either halothane or enflurane [15, 21], whilst their effects in

patients with pulmonary hypertension is not known. Isoflurane decreased PVR and PAP when used during open lung biopsy in a patient with primary pulmonary hypertension [22]. Nitrous oxide was shown to cause considerable increases in PVR in patients with pulmonary hypertension, even when administered with halothane or fentanyl [23]. However, in another study of patients with only moderate pulmonary hypertension (mean 37 mmHg, SD/14 mmHg) undergoing mitral valve replacement, nitrous oxide caused a slight decrease in PAP [24].

There is little information about the effects of intravenous induction agents on the pulmonary circulation in pulmonary hypertension. However, most agents, with the possible exception of ketamine which may increase PVR [25], have either little effect on PVR or decrease it. Similarly, modern opioids have little effect on PVR [26] whilst the use of an alfentanil infusion in severe pulmonary hypertension was found to be beneficial [27]. High dose opioid anaesthesia, using fentanyl or sufentanil, resulted in no change in PVR or PAP [28].

A stress-free anaesthetic with controlled ventilation, gentle intubation and continuous haemodynamic monitoring would appear to provide ideal conditions. Regional techniques may be equally satisfactory. Epidural anaesthesia has been used for Caesarean section [29] and for the management of labour [20, 30].

Thus, thoracic epidural analgesia should provide good intra-operative conditions for thoracic surgery, since it reduces the stress of the surgery and allows controlled ventilation. It has the additional advantage of providing good postoperative analgesia. In this patient, it resulted in a reduction in PAP throughout surgery and in the first 24 h of recovery. Following the initial dose of bupivacaine, the PVR decreased slightly. After induction there was an increase in PVR although PAP remained at less than its pre-anaesthetic level and throughout the operation stayed relatively constant. There was little effect on the systemic blood pressure, although a decrease in cardiac output was compensated for by a rise in SVR. Moreover, in the first 24 h postoperatively the pulmonary circulation appeared stable, with no increase in either PAP or PVR.

In summary, the use of thoracic epidural analgesia for a patient with severe primary pulmonary hypertension undergoing lung biopsy appeared to help to maintain a stable pulmonary circulation, without adverse effects on the systemic pressures. Postoperatively, an infusion of a mixture of bupivacaine and fentanyl maintained this stability.

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Functionally crossed pipelines

An intermittent condition caused by a faulty ventilator

W. M. WEIGHTMAN, V. FENTON-MAY, R. SAUNDERS, A. LEWIS AND C. C. WISE

Summary

Unexpectedly high blood oxygen tension in intensive care patients occurring over a 5-year period were found to have been caused by an apparently normally functioning ventilator which was intermittently acting as a conduit between the oxygen and air pipelines. Responses to a circular letter sent to Quality Control Pharmacists in England and Wales suggest that similar faults may have occurred at least five times in the past 10 years. Recommendations are given for the prevention of this problem, including monitoring of gas pipeline composition.

Key words

*Medical gases, pipeline; contamination.
Ventilator; malfunction.*

Over a period of 5 years, arterial blood gas analysis results with unexpectedly high oxygen tensions were occasionally recorded from patients in the intensive care unit (ICU) at the Cardiff Royal Infirmary. Various causes for these abnormal results were postulated. On several occasions ventilators were taken out of service as they appeared to be delivering 100% oxygen, but on examination and testing were subsequently proven to be delivering accurately the dialled concentration of oxygen. Investigation of these episodes of hyperoxia was difficult as they occurred mainly at night and at weekends, but eventually, during one of the episodes, 100% oxygen was proven to be coming from all the compressed medical air pipeline outlets in the intensive care unit; between 100–200 l of gas issued from the air pipeline before air, rather than oxygen, appeared.

Pipeline system

The ICU pipeline system had been installed 8 years earlier. The compressed medical air system consists of two compressors: one working, the other in reserve, feeding separate dryers and reservoirs. There are two separate outlets from each reservoir, regulated at 4 bar (400 kPa) and 7 bar (700 kPa). The 7 bar system supplies the two orthopaedic operating theatres and is used only for surgical instruments, whilst the 4 bar system supplies respiratory air

to the orthopaedic operating theatres, intensive care unit, accident and emergency department, and one ward. The oxygen system consists of a vacuum insulated evaporator (VIE) which supplies all the wards and operating theatres of the main hospital building and is regulated to 4.2 bar. Cylinder backups exist for both air and oxygen systems but neither backup was in use during the episodes described above.

Contamination of the air supply was finally tracked down to a Bourne Bear 1 ventilator which was acting as an intermittent conduit between the oxygen and air pipelines. The ventilator appeared to function normally and had undergone an annual maintenance service, carried out to the manufacturer's specifications, during each of the 5 years in question. However, both in the intensive care unit, and when connected to a test rig in the Respiratory and Anaesthetic Support Services Department, the ventilator could repeatedly be provoked to pass up to 50 l.min⁻¹ of oxygen into the air pipeline. This occurred while the electrical power to the ventilator was turned off, on approximately 5% of occasions that the machine was cycled through a test procedure, and required air consumption elsewhere on the pipeline. Only a slightly louder than normal hissing sound, audible at the back of the machine, betrayed the passage of large quantities of gases between the two pipelines. No gas issued from either hose when

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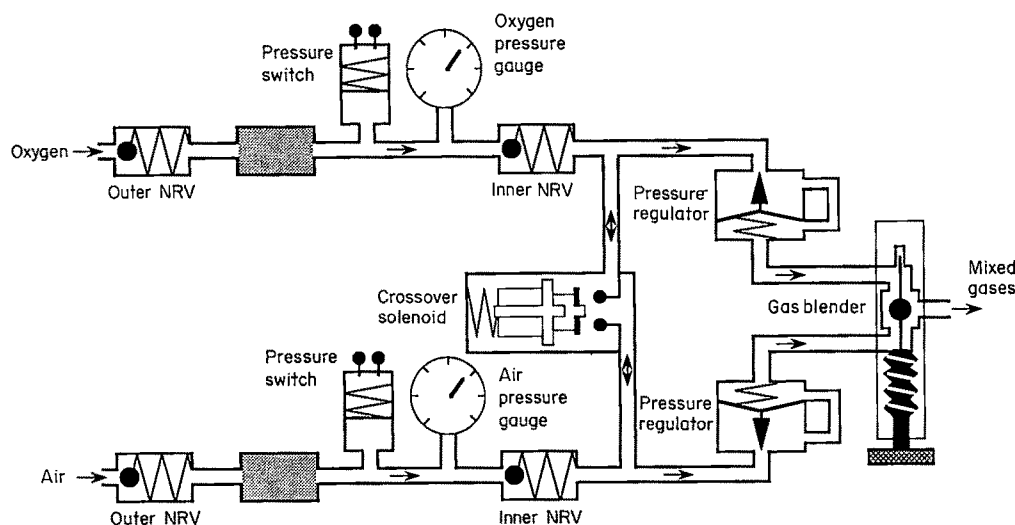


Fig. 1. Simplified diagram of the gas inlet of the Bear 1 ventilator. (NRV = nonreturn valve)

singly disconnected from the pipeline. Restarting the machine, disconnecting it from the air supply, or stopping concurrent use of the air pipeline, terminated the fault.

Examination of the pneumatic diagram for the ventilator (Fig. 1) suggests the cause of the fault. Air and oxygen supplies are connected at pipeline pressure via an electrically operated valve, the 'cross-over' solenoid. This feature is present so that in the event of one gas failing the 'cross-over' solenoid will open and allow the remaining gas to supply both sides of the air/oxygen mixer, in an attempt to keep the ventilator performance unchanged in all but its oxygen delivery concentrations. The 'cross-over' solenoid is open when the electricity to the ventilator is off, but backflow of gas is prevented by two nonreturn valves in each supply line. We suggest, but have been unable to prove, that the cause of the problem which we experienced was intermittent failure of the nonreturn valves in the air supply line; both of these nonreturn valves are based upon ball bearings, one of the valves was a sealed unit and could not be dismantled for inspection, the other did not show any clear macroscopic cause for intermittent failure.

Discussion

This report highlights an unusual failure of a piped medical gas system which occurred intermittently over a period of 5 years, despite full accord with the standards laid down by the Department of Health in Health Technical Memorandum No. 22 (H.T.M. 22) [1].

Much design, engineering, and quality control effort is expended to prevent failure of pipeline gas supplies. H.T.M. 22 specifies that particular attention must be paid to preserving and monitoring gas pressure; there must be reserve gas supplies with an alarm which activates when the reserve is in use, and also an alarm which activates when the gas pressure falls below specified limits. Alarm repeaters must be placed in specified locations so that both users and maintainers of a gas supply will be warned of problems at all times of the day.

In contrast to this, H.T.M. 22 specifies that identity and composition checks of the pipeline contents should only be carried out when a system is commissioned or has high

hazard work performed. Thus contamination of pipeline gas by equipment subsequently connected to it will always remain undetected until it causes a clinical problem. There have been several instances, in addition to the present report, where this has happened. In the USA a fault in the air flowmeter of an Emerson IMV ventilator used in an adult ICU resulted in oxygen displacing air from the compressed air supply to a neonatal intensive care unit [2]. In Switzerland, a faulty Bird oxygen blender connected to 4.1 bar oxygen and 8 bar compressed air, resulted in air entirely displacing oxygen from the oxygen pipeline [3]. A similar problem occurred in Glasgow where 5.4 bar air displaced oxygen from a 4.1 bar oxygen pipeline, and was thought to have been caused by a faulty mixer valve attached to a Siemens Servo 900 Ventilator [4].

When the manufacturers and the Department of Health were notified of this problem, it became apparent that similar malfunctions had occurred before but a fault in the nonreturn valve had not been proven. Both said they were seeking a test for detecting the problem.

Further evidence of such occurrences was obtained when we sent a letter to quality control pharmacists in England and Wales which uncovered five more incidents of unexplained gas pipeline contamination. Three of these incidences were of air replacing oxygen, one of nitrous oxide replacing oxygen, and one of oxygen replacing air, and in each instance malfunctioning respiratory equipment was suspected.

An increasing amount of equipment is connected to hospital gas pipelines, some of it of dubious age and quality. We speculate that a tragedy will occur if steps are not taken by hospitals, manufacturers of respiratory equipment, and suppliers of medical gases, to prevent this from recurring. We recommend, in addition to the present safeguards: (1) the composition of pipeline gases should be monitored at the terminal end of each branch of the system. An audible and visual alarm should be activated if gas quality varies; (2) oxygen should always be kept at the highest pipeline pressure of all the respirable gases in use. Air at higher pressure should only be used for powering surgical tools; (3) staff using respiratory equipment should remember that the pipeline system is not infallible; cross-

over, contamination, and supply failure can happen despite the many safety checks employed. This should be considered when teaching the management of unexpected hypoxia. A cylinder of oxygen with a self inflating resuscitation bag should be available in all locations where patients' lungs are ventilated; (4) where possible, equipment should be designed so that communication between gases only occurs after they are regulated to a lower pressure. Defective check valves have been responsible for two of the three previously reported incidences of pipeline contamination and should not be relied upon to prevent backflow from the machine into the pipeline.

In addition we would like to suggest two 'universal precautions' that all staff should adopt: (1) always use an oxygen analyser on anaesthetic machines and intensive care

ventilators; (2) always disconnect respiratory equipment from the pipeline supply when not in use.

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Spinal epidural anaesthesia

A new combination system

R. LIFSCHITZ AND R. JEDEIKIN

Summary

We present our experience with a new combined spinal epidural system through which regional anaesthesia was performed in 30 male patients undergoing suprapubic prostatectomy. The technique consists of two needles, a 17 G Tuohy needle with a hole in its distal curve (back eye) and a 29 G spinal needle which is passed through the back eye before being introduced into the subarachnoid space. We found the back eye combined spinal–epidural system effective and simple to use. The Tuohy needle with its back eye did not impede insertion of the epidural catheter and was a suitable introducer for the thin 29 G spinal needle.

Key words

*Anaesthetic techniques, regional; spinal, epidural.
Equipment; needles.*

The technique of combining spinal with epidural anaesthesia (CSE) is a recent innovation of regional anaesthesia by which the main advantages of subarachnoid and epidural anaesthesia are retained and combined [1–3]. Spinal anaesthesia offers rapid onset of action, reliable surgical anaesthesia and good muscle relaxation, while the insertion of a catheter into the epidural space enables smaller amounts of anaesthetic agent to be used and reserves the option of extending the block for surgery or topping up with an opioid for postoperative analgesia.

Initially, separate intervertebral spaces were used for CSE [1]. This technique has subsequently been replaced by a single level approach whereby, after placing the Tuohy needle in the epidural space, a spinal needle (usually 26 G) is threaded through the larger bore epidural needle into the subarachnoid space [2, 3]. More recently a combined needle was made by welding a spinal needle onto the side of a Tuohy needle [4]. Potential dangers of these various systems include: breaking off the distal part of the brazed spinal needle from the Tuohy needle; shearing off the distal end of the spinal needle as it is manoeuvred through the Huber tip of the Tuohy needle; inserting the epidural catheter into the subarachnoid space through the dural hole made by the spinal needle. Turning the Tuohy needle through 180° to avoid this latter complication has been advocated but this technique is in itself a common cause of accidental dura puncture and postspinal headache.

A new CSE system (Mediziv Medical Products, Israel) has been introduced in an effort to eliminate some of these problems. The Tuohy needle is manufactured with a hole (back eye) in its curve so that the spinal needle, instead of exiting from the end of the Tuohy, passes through the back eye directly into the subarachnoid space (Figs. 1 and 2).

The spinal needle fits snugly into the hub of the Tuohy needle, and when inserted to the hilt protrudes 0.75 cm beyond the end of the Tuohy needle. We report our experience of a pilot study in which regional anaesthesia was performed in 30 patients using the combination of a back eye Tuohy needle and a 29 gauge spinal needle.

Methods

Thirty males, aged 59 to 80 years old, scheduled for suprapubic prostatectomy were studied; patients who suffered from headache or backache were not studied. Informed consent was obtained from all patients. Premedication consisted of 2–3 mg midazolam given intramuscularly 30–45 min before anaesthesia and was followed by an intravenous infusion of 750–1000 ml of compound sodium lactate solution. Patients were turned onto their sides and a 17 G × 80 mm back eye Tuohy needle advanced, with the bevel of the needle directed cephalad, into the epidural space at the L₃₋₄ or L₄₋₅ interspace. A 29 G spinal needle, 0.75 cm longer than the Tuohy needle, was then passed

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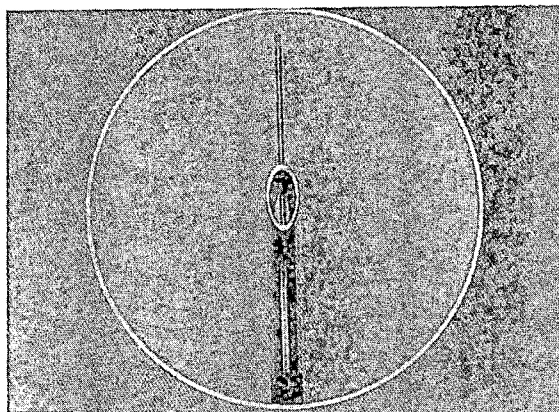


Fig. 1. Close up of the 29 G spinal needle protruding through back eye of the Tuohy needle.

through the back eye of the Tuohy needle. Correct placement of the spinal needle in the subarachnoid space was confirmed by aspiration of cerebrospinal fluid (CSF). After the subarachnoid injection of 2 ml of 0.5% hyperbaric bupivacaine, the spinal needle was removed and a catheter introduced into the epidural space. Ten ml of 2% lignocaine was then injected through the epidural catheter. Approximately 15 min before the end of surgery 3 mg of morphine was injected through the epidural catheter for postoperative analgesia. Further increments of epidural morphine were injected as required. On the fourth postoperative day the epidural catheter was removed.

The following parameters were evaluated in all patients: level of sensory block measured by pin prick, ease and success of insertion of the spinal needle through the Tuohy back eye, and complications (headache and backache). Surgical anaesthesia was assessed by the surgeon and graded poor, fair or good. Analgesia was considered good when no further supplementary drugs were given. Arterial blood pressure, heart rate, electrocardiogram changes, percutaneous arterial oxygen saturation and respiratory rate were monitored. Hypotension was defined as a 20% fall in blood pressure below the pre-induction level or a systolic blood pressure below 100 mmHg.

Results

Surgical analgesia was considered good in all 30 patients. The level of sensory block was between T₆ and T₁₀ and no patients required supplementary analgesia or sedation.

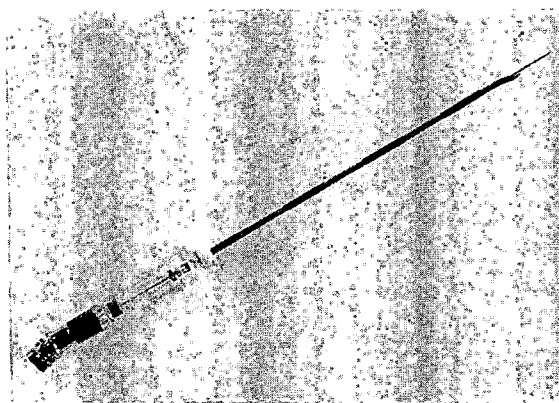


Fig. 2. Lateral view of the 29 G spinal needle protruding through the back eye of the Tuohy needle.

Hypotension occurred in seven patients and was corrected immediately by giving intravenous ephedrine (5 mg increments) and fluids. Before each CSE injection the system was checked by passing the spinal needle through the Tuohy needle. In all 30 cases the spinal needle was noted to pass straight through the back eye of the Tuohy and on no occasion did it ever bypass the back eye and exit through the Huber point.

On performing the CSE injection, the spinal needle was introduced into the subarachnoid space with no difficulty in 29 of the 30 patients. In one patient blood was aspirated and blocked the needle, requiring a change of CSE needles and intervertebral space. Because of the small size of the spinal needle, and in order to confirm subarachnoid placement, CSF was actively aspirated with a 5 ml plastic syringe.

There were no cases of headache or neurological sequelae; one patient complained of mild backache which lasted for 12 h. No respiratory difficulty or depression was noted.

Discussion

The proclaimed benefits of CSE [1–4] were also demonstrated in our study. All patients had good analgesia, full muscle relaxation and good postoperative pain relief. We also found the back eye CSE system very easy to use. Once the Tuohy needle was positioned in the epidural space, the spinal needle was rapidly and successfully inserted into the subarachnoid space, the only failure being due to a blocked needle.

The problems encountered in using this CSE system are for the most part due to the small size of the spinal needle. CSF does not flow spontaneously through a 29 G needle and active aspiration is required to confirm placement in the subarachnoid space. A bloody tap may also block the 29 G needle requiring a change of the set and intervertebral space.

The most positive aspect of the CSE is the ease with which the spinal needle enters the subarachnoid space. In essence the Tuohy needle is a perfect introducer for the thin spinal needle. Furthermore, the ability to introduce an epidural catheter for intra-operative and postoperative analgesia without the trauma of having to insert a second needle into the patient's back is a most attractive option. An additional positive feature of the back eye CSE set is the exact fit of the spinal needle into the hub of the Tuohy needle, facilitating the injection of local anaesthetic through the spinal needle. Nickalls and Dennison, using a much longer spinal than Tuohy needle, reported a high incidence of failed spinal anaesthesia thought to be due to inadvertent malpositioning of the tip of the spinal needle outside the dura while injecting [5]. They attempted to overcome this problem by clamping the spinal needle with an artery clip at the point where it enters the Tuohy needle. However, this modification may damage the needle and obstruct fluid flow, while the weight of the clip may drag the spinal needle out of the subarachnoid space.

Although we used a 29 G spinal needle in our study, 26 G needles of the same length and type are also produced and pass readily through the back eye of the 17 G Tuohy needle. However, since the incidence of spinal headaches using 29 G spinal needles is very low [6] and the 26 G needles available in these CSE sets are not pencil point [7], we elected to use 29 G and not 26 G needles in our patients.

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Modification to the Sprotte spinal needle

M. Y. AGLAN AND P. K. STANSBY

Summary

The large lateral hole of the Sprotte needle is claimed to aid cerebrospinal fluid efflux during the performance of spinal anaesthesia. Using theoretical calculations and practical measurement we have shown that reducing the area of the lateral hole to that of the cross sectional area of the needle does not affect the flow rate. We suggest this modified Sprotte needle is an improved design which has the potential advantages of reducing the incidence of inadequate spinal anaesthesia and strengthening the needle tip.

Key words

Equipment; needle, spinal.

Spinal anaesthesia is undergoing a resurgence of interest as a technique for many surgical procedures; however, post-lumbar puncture headache (PLPH) still remains a problem associated with its use. A new atraumatic spinal needle (the Sprotte needle) has been reported to reduce markedly the incidence of PLPH [1, 2] and is said to have the added advantage of easy injection and aspiration due to the relatively wide lateral hole. We are unaware of any evidence to support the benefit of a large lateral hole; assuming laminar flow, the ideal design of a needle should have a lateral opening equal to the internal cross-sectional area of the shaft.

To investigate the effect of a large lateral hole we measured the size of the lateral hole and the cross-sectional area of the 24 g Sprotte-PAJUNK needle and calculated the Reynolds number to indicate the nature of the flow through the needle. We then adapted the needle so as to reduce the size of the lateral opening to be nearly equal to the cross-sectional area of the shaft of the needle and measured the flow through both the original and the modified Sprotte needle.

Measurements and calculations

Measurements

The internal diameter of the needle (ID) was measured by trial with wires of circular cross section. The wire's diameter was measured by a micrometer. The ID was found to be 0.35 mm.

The length and width of the lateral opening (Fig. 1) were measured by optical comparison with standard engraved

scales under 10 times magnification. The length of the lateral opening (A) was 1.7 mm, and its width (B) was 0.32 mm.

So, the area of the lateral opening (considered as a rectangular) = $A \times B = 1.7 \times 0.32 = 0.544 \text{ mm}^2$.

The cross-sectional area of the needle = $\pi \times r^2 = 3.142 \times (0.35/2)^2 = 0.096 \text{ mm}^2$.

Calculation of fluid velocity and Reynolds number (R)

The flow of cerebrospinal fluid (CSF) through the needle is due to the pressure or 'head' difference between the inside of the body and the outside. CSF will pass first through the hole then along the length of the needle.

For laminar flow, the formula for head loss (Hp) along the tube is given by

$$H_p = 32 \mu L V / \delta g D^2 \quad (\text{eqn 1})$$

where ' μ ' is viscosity of CSF (1.006 centipoise or mPa.s), ' L ' is tube length (93 mm), ' V ' is average flow velocity, ' δ ' is CSF density (approximately 1000 kg.m^{-3}), ' g ' is gravitational acceleration (9.81 m.s^{-2}), ' D ' is internal diameter of the needle (0.35 mm).

For this formula to be valid for laminar flow, the Reynolds number (R) must be less than 2000.

$$R = \delta V D / \mu \quad (\text{eqn 2})$$

The formula for 'head' loss through the hole (Hh) is given by;

$$H_h = K v^2 / 2 g \quad (\text{eqn 3})$$

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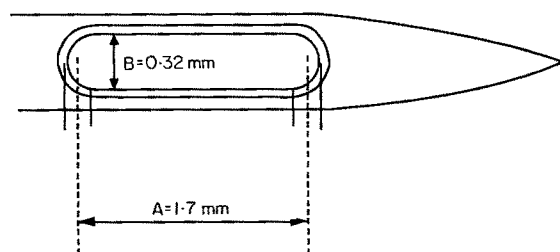


Fig. 1. Measurement of the lateral hole of the Sprotte needle.

where 'K' is a constant of order 1 and 'v' is the velocity through the hole [3].

Considering a pressure head of 120 mm (which is the average normal CSF pressure with the patient lying in the lateral position) [4], and substitution in equation 1, a flow velocity of 0.05 m.s^{-1} is obtained. This gives a Reynolds number of 17.5 (confirming laminar flow). Considering a pressure head of 450 mm (which is the average normal CSF pressure with the patient in the sitting position) [4], a flow velocity of 0.18 m.s^{-1} is obtained. This in turn gives a Reynolds number of 63 (still confirming laminar flow).

The value of head drop across the hole (equation 3), assuming the hole to be of the same diameter as the needle is 0.1 mm (for a pressure head of 120 mm) and 1.6 mm (for a pressure head of 450 mm). This is negligible in relation to the overall pressure 'head' difference.

Flows through the original and modified 24 g Sprotte needle

We reduced the size of the lateral hole by passing the best fit wire for the needle shaft through the hole. The rest of the hole was sealed with a metallic glue. A simple experiment was set up (Fig. 2).

Normal saline, which has the same viscosity as CSF, was allowed to drip through the needle into a container over a period of 4–5 min, using an average head of 120 mm. The container was then weighed and the flow calculated. This was repeated three times using three different needles of each type (original and modified Sprotte).

In all cases the flow was $0.0100 \pm 0.0001 \text{ ml.s}^{-1}$. The experiment was repeated with a head of 450 mm giving a flow rate of $0.024 \pm 0.001 \text{ ml.s}^{-1}$.

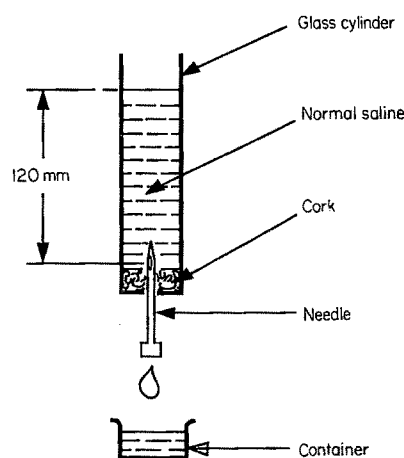


Fig. 2. Experimental test rig used to measure flow through the needle.

Discussion

CSF coming out of the body is driven by a CSF pressure above atmospheric pressure. The driving pressure head is 'absorbed' by the head required to overcome friction along the tube and that required to 'push' CSF through the hole. The latter is usually referred to as minor head loss since it is generally small in relation to the head loss due to friction along a tube and this is confirmed in this case by our calculations.

Calculating Reynolds number, with pressure heads of 120 and 450 mm, we obtained figures of 17.5 and 63 respectively. These numbers are well below the generally accepted limit of 2000 and confirm laminar flow through the needle. As the diameter of the needle is the most important factor affecting the flow, there is no benefit in having a lateral hole area larger than the cross-sectional area of the needle. From our measurements, the area of the lateral hole is more than five times greater than the cross-sectional area of the needle.

There was no difference in the measured flow through the original and the modified Sprotte needle. This indicates that the lateral hole size may be reduced from 1.7 mm in length to 0.32 mm without impeding the fluid flow in any noticeable way. This reduction in the length of the hole may also improve the structural stability of the needle's tip.

Failed and incomplete spinal anaesthesia has occurred using Sprotte needles, and postulated as due to some of the local anaesthetic being injected outside the subarachnoid space because of the long lateral hole [2, 5]. This problem can be avoided by pushing the needle 1–2 mm inward after the CSF has appeared. However, this may cause unnecessary trauma to the cauda equina. Reducing the length of the hole would reduce the likelihood of this problem.

We believe that the easy injection and rapid efflux of CSF seen with Sprotte needles and claimed to be due to the relatively large lateral hole, is actually related to the relatively broader gauge of the needle itself. We suggest that a modified Sprotte needle having a lateral opening area equal to the internal cross-sectional area will not alter the flow characteristics and may have the potential advantages of adding more strength to the needle tip and minimising the possibility of inadequate spinal anaesthesia.

Acknowledgment

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Magnetic resonance for the anaesthetist

Part II: anaesthesia and monitoring in MR units

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Summary

Anaesthetists are increasingly involved in patient care during magnetic resonance imaging and spectroscopy. This paper describes a system which has been developed for the management of critically ill patients and the conduct of anaesthesia in a magnetic resonance unit with a 1.6 tesla whole body magnet. Difficulties which arise from working in a confined space in a high magnetic field are highlighted. Different approaches to anaesthesia, sedation and the modification of equipment for use in this environment are reviewed. The problems associated with patient monitoring within a magnetic field are discussed and some solutions are suggested. A transport system for critically ill patients is described and a protocol for management is outlined.

Key words

Measurement techniques; nuclear magnetic resonance.

Anaesthesia; magnetic resonance imaging.

Monitoring; blood pressure, carbon dioxide; electrocardiography, oxygen.

Equipment; anaesthesia machines.

Magnetic resonance imaging (MRI) is now established as a valuable imaging technique, particularly for the investigation of the central nervous system. Magnetic resonance spectroscopy (MRS), which is based on the same physical principles as MRI, is used to provide biochemical information from a variety of different nuclei (^1H , ^{19}F , ^{31}P , ^{13}C) [1]. The number of MR systems is increasing and new applications for the technique are still being developed; in 1990 approximately 100 000 examinations were performed in the UK. The requirement for anaesthetists to provide sedation or anaesthesia and to manage critically ill patients in the difficult environment of a magnetic field is also likely to increase.

In this paper we discuss the problems of administering anaesthesia for MRI and review the existing literature on the subject. On the basis of our experiences of MRI and MRS in a 1.6 tesla (T) magnet system, we suggest how these problems may be solved using, wherever possible, commercially available equipment.

General considerations

Indications for anaesthesia and sedation (Table 1)

The need for immobility. The images and spectra are composed of multiple data acquisitions which are averaged

by the system computer to give the final image or spectrum. At present, some individual scans may take up to 20 min and the whole examination may last over an hour. Movement of the patient at any point during the multiple acquisitions which make up one scan will result in faulty spatial allocation of signal and so degrade the quality of the final image by the production of movement artifacts. In addition, if the patient changes position at any time during the examination the homogeneity of the magnetic field, which has been maximised to his original position, is reduced and this may also degrade the quality of all the subsequent scans obtained. This factor is of more importance in MRS than during an MRI examination.

Patient anxiety. Severe anxiety may be a significant problem for which the examination may have to be aborted. The confined space, the loud noises which occur during the scans, the temperature of the magnet bore and the length of the procedure were found to be the principle causes of distress [2]. Flaherty and Hoskinson [3] investigated 210 patients undergoing MRI and found that 4% became too anxious to complete the procedure. Sedation or anaesthesia is an extreme solution to this particular problem. Education and counselling, the presence of a companion, gradual familiarisation with the enclosed space and prone positioning [4] should be tried first.

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Table 1. Indications for sedation or general anaesthesia for a magnetic resonance examination.

Indication	Patient group	Action
Reduce patient movement	Infants and small children. Confused, intellectually subnormal, mentally ill adults.	Sedation or anaesthesia.
Airway protection	Unconscious patients. Critically ill patients receiving IPPV.	Anaesthesia Anaesthesia and IPPV
Control of ventilation	Head injuries. Patients with raised ICP where P_{aCO_2} has to be controlled.	Anaesthesia and IPPV
Anxiety and claustrophobia	Adults.	Reassurance, education, counselling, prone position. Sedation. Anaesthesia only if necessary and examination essential.

IPPV, intermittent positive pressure ventilation.

ICP, intracranial pressure.

Table 1 summarises the types of patients who may require anaesthesia or sedation in order to undergo MR examination.

Problems of anaesthesia in the MR environment

Anaesthesia and monitoring of patients in an MR system present several problems which have been discussed in detail elsewhere [1]. In summary, these are: (1) the need to exclude ferromagnetic equipment from the magnetic field; (2) limited access to and view of the patient within the magnet bore; (3) malfunction of monitoring equipment, or interference, produced by the changing magnetic field gradients and rapidly switching radiofrequency currents; (4) degradation of image and spectral quality arising from stray radiofrequency currents produced by monitoring equipment and leads.

Location of the anaesthetic equipment

The principal decision to be made by the anaesthetist concerns the location of the anaesthetic machine, ventilator and monitoring equipment in relation to the magnet. This will depend upon the strength of the magnetic field and the type of magnetic field shielding in use [1].

At present, however, there are two possible options, the choice of which may be dictated by the layout of the magnet suite.

Outside the magnetic field. All anaesthetic and monitoring equipment may be kept outside the magnetic field (or Gauss) line at which attraction of ferromagnetic items is likely to occur. This is normally between the 30 and 50 Gauss lines. The distance of these lines from the centre of the magnet depends on field strength and shielding [1]. If the anaesthetic machine, monitoring equipment and ventilator are sited several metres from the magnet face, long monitoring leads and ventilation tubing, with a large compressible volume in the circuit, are required. There is an increased risk of disconnection and ideally two anaesthetists should be present, one by the magnet bore close to the patient and the other to observe the equipment. If two anaesthetists are not available, the second person should be an appropriately trained anaesthetic assistant.

Close to the magnet. The anaesthetic and monitoring equipment may be sited close to the magnet bore, in which case the anaesthetic machine and its components must be non-ferromagnetic. Monitoring equipment powered by an unfiltered mains supply may introduce interference. If battery powered units are used they should be in a permanently fixed, secure position since batteries are strongly magnetic. Once the examination has started, the position of the anaesthetic machine should not be changed because the repositioning of a large metallic mass may degrade magnetic field homogeneity.

New developments in magnet design are likely to help the anaesthetist in future. The trend in imaging is to move to lower field strengths. Open magnets are being built, in which the patient is placed between pillars within the field, rather than into the enclosed tube of a magnet bore. In these open systems, access, visualisation and monitoring of the patient will be greatly improved.

Review of existing literature

Authors from a number of MRI centres have described various modifications to existing anaesthetic equipment. The first report of anaesthesia in an MR scanner was from Geiger and Cascorbi [5] who sited the anaesthetic machine in the MR control room distant from the magnet and allowed the patient to breathe spontaneously while anaesthesia was maintained by the intravenous route. A simple breathing system for manual ventilation of children, composed of corrugated tubing up to 16 m long, connected to a Laerdal self-inflating bag with a nonrebreathing valve, was described by Liao *et al.* [6]. Boutros and Pavlicek reported the use of a Mapleson D system, with oxygen delivered from 6 m away by several connected tubes [7]; co-axial Mapleson D circuits have been used elsewhere [8,9]. Neumark *et al.* used a Siemens Servo 900-D ventilator placed more than 7 m away, in both adults and children and employed nomograms to calculate the volume of compressed gas lost in the 9 m long tubing [10].

Mirvis *et al.* used an unmodified Siemens Servo 900-C ventilator, placed 1.2 m from the bore of a 1.5 T magnet, for both adults and children. The positive end-expiratory pressure (PEEP) valve of the Servo 900-C is of a solenoidal

type which may be affected by the magnetic field and can malfunction if placed closer than this to the magnet [11].

Cady *et al.*, who were investigating birth asphyxiated infants, modified a conventional transport incubator and its ventilator to stand 3 m away from a 1.9 T magnet [12]. McGowan and Erenberg ventilated neonates with a modified Bio-Med Devices MVP-1C ventilator which was sited 3 m away in a 0.5 T magnet [13].

Prien *et al.* used a commercial anaesthetic machine and breathing system with minor modifications, situated close to the magnet bore [14].

Rejger *et al.* sited their anaesthetic machine close to the 100 Gauss line and ventilated the patient through a specially adapted breathing system attached to a Dräger Oxylog ventilator which has no ferromagnetic components [15].

Dunn *et al.* used a fluidic ventilator close to the magnet bore to passively ventilate the lungs of volunteers [16]. This ventilator was subsequently modified to become the MRI compatible (Monaghan Medical Corp., Plattsburg, NY) which Smith and colleagues used in conjunction with anaesthesia maintained using a total intravenous technique [17]. This ventilator has also been used in other centres [18–20]. Barnett *et al.* incorporated a Puritan-Bennet ventilatory pressure alarm [19] which has magnetic components, therefore the ventilator was sited outside the magnet room. This modified ventilator was also used by Karlik *et al.*, who, in addition, modified an Ohmeda anaesthetic machine. Magnetic parts were replaced with nonmagnetic grade stainless steel and ferromagnetic oxygen cylinders with aluminium ones [20].

A modified Foregger Model BC anaesthetic machine has also been used within 1 m of a 1.5 T magnet [21]. Ferromagnetic castors and support struts were replaced with stainless steel and aluminium counterparts and again, aluminium cylinders were used.

A fully integrated ventilatory and monitoring system, complete within its own Faraday shield to reduce radiofrequency electromagnetic emission, for use with a 2.35 T magnet was described by Boesch *et al.* [22,23].

Current management of anaesthesia and sedation

We have experiences of imaging patients at two different magnetic field strengths, 0.15 T and 1.6 T. The latter system is a prototype magnet which is used principally for spectroscopy research, and our practice has been developed mainly around this system. This account is intended to assist those anaesthetists who wish to purchase or modify equipment for regular use in an MR unit. However, patient management in a situation in which no specialised equipment is available is also described.

Anaesthetic machine and mechanical ventilator

We have sited the anaesthetic machine and monitoring equipment between the 5 Gauss and 30 Gauss lines (8–9 m from the patient's head) because of the design of this prototype 1.6 T magnet. We use an unmodified Excel 210 (Ohmeda: Hatfield, UK) anaesthetic machine which is supplied by a piped gas installation. Ohmeda now make a modified version of the Ohmeda XL 210 for MRI use which is nonmagnetic. It is 99.1% nonmagnetic by weight, has aluminium cylinders and includes an MRI compatible oxygen monitor. It has been tested with a 1.5 T system at a

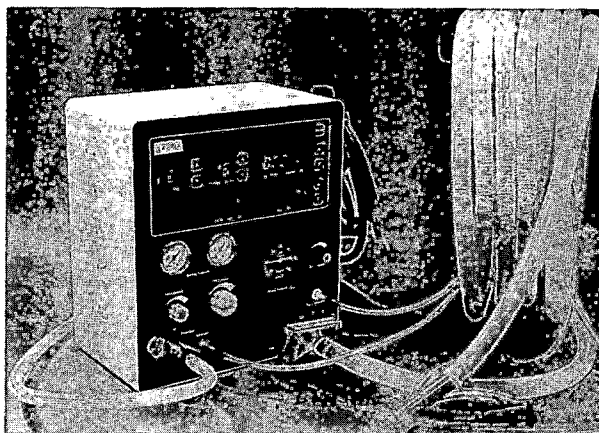


Fig. 1. The CW 200A ventilator with extended disposable tubing.

field of approximately 2300 G and its performance, and that of the Fluotec 4 vaporizers, were within normal limits [24,25]. No degradation of image quality was noted. The system, which costs about £9500 (May 1991), is now available in the UK. However, at present, aluminium gas cylinders are not readily available in the UK (written communication to Ohmeda/BOC March 1991). Penlon, PneuPac (Luton, Bedfordshire, England) and Siare Hospital (S.R.L., Bologna, Italy) are also manufacturing nonmagnetic anaesthetic apparatus.

We have used either a Penlon Nuffield Series 200 or the new CW 200 ventilator [26] (Fig. 1). Both ventilators are suitable for adults, children and neonates (with the addition of a Newton valve to the Nuffield 200). The Nuffield 200 behaves as a pressure generator [27], therefore any reduction in compliance or increase in resistance at the patient end of the system leads to a reduction in ventilatory volume. This is of particular concern in the MR environment when the patient's chest wall movement is not visible, and very long breathing circuits are being used. We found that the maximum mouth pressure generated by the Nuffield 200 attached to a 10 m long Bain system was 45 cmH₂O. Since the safety valve blows off at pressures above this, the system is unsuitable for use in adults with noncompliant lungs. The CW 200 has the advantage of being a valveless ventilator, therefore if the casing was plastic, it could be taken safely to the edge of the magnet bore. The CW 200 has the characteristics of a pressure generator, but with features which increase its safety. Tidal and minute volumes are continuously displayed and calculated from the expired volume and the machine maintains a constant volume using a servo system. Variable PEEP can be applied without the addition of a separate valve, therefore it is more suitable for use in critically ill patients in whom PEEP is required. Hatch *et al.* [28] compared the Nuffield 200 and the CW 200 for use with a T-piece in paediatric anaesthesia. They found that the CW 200 caused a greater reduction in end-tidal CO₂ values at the same tidal volumes as the Nuffield 200. This may have the additional advantage of avoiding barotrauma [26].

Breathing systems

We have used a noncoaxial Mapleson D system, and a coaxial Mapleson D (Bain) system, both of which were approximately 10 m long. The extended Bain system

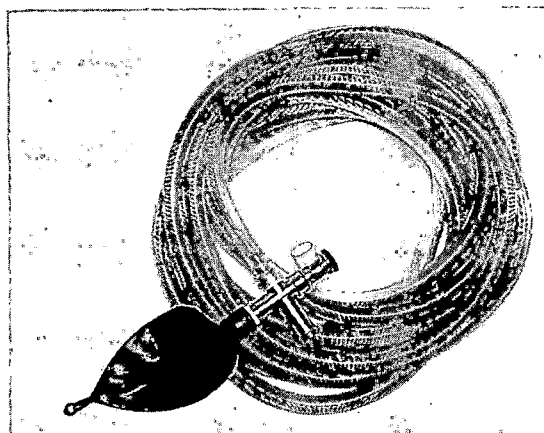


Fig. 2. A coaxial Mapleson D (Bain) breathing system, 10 m long.

(Fig. 2) was supplied by the manufacturer (Penlon Limited, Abingdon, UK). Concern has been expressed about increased expiratory resistance with the Bain system [18]. We measured expiratory pressures both in the noncoaxial Mapleson D system and the Bain system and found them to be $0.33 \text{ cmH}_2\text{O.m}^{-1}$ and $0.5 \text{ cmH}_2\text{O.m}^{-1}$ respectively. Therefore, at a length of 10 m an extra $1.7 \text{ cmH}_2\text{O}$ expiratory resistance is generated by the Bain system. When the Heidbrink valve was included in the Bain system the expiratory resistance rose to $0.6 \text{ cmH}_2\text{O.m}^{-1}$. These pressures were measured at a flow of 60 l.min^{-1} which can be achieved in critically ill patients with high minute volume requirements. We have used the Bain system only in conjunction with the Penlon-Nuffield 200 ventilator to ventilate adults.

A modified T-piece system will permit either spontaneous respiration or manual ventilation in adults. This may be the method of choice when the environment of an



Fig. 3. A patient with the head enclosed by a receive coil. Little space is available for a standard tracheal tube and catheter mount, a RAE tube minimises this problem and facilitates placement of anaesthetic tubing.

MR unit is unfamiliar and no provision has been made for anaesthesia (Table 2). A T-piece system and a length of disposable oxygen tubing can be attached to the fresh gas outlet of an anaesthetic machine sited well outside the 50 Gauss line. We have had no problems with lengths up to 10 m and at this distance from the magnet oxygen cylinders can be changed safely if piped gases are unavailable. Further management is discussed elsewhere in the text. Other centres have used an extended Bain system [8,9]. Both the coaxial Mapleson D and noncoaxial Mapleson D breathing systems are cumbersome to store and, because of the long lengths of tubing, should be meticulously checked before use.

Airway management

We consider that the airways of anaesthetised or unconscious individuals should be maintained with a tracheal tube since the patient is not visible in the magnet bore and the head is inaccessible. The use of a RAE tube facilitates the placement of ventilation tubing [18]. In some magnet systems, in which a receive coil encloses the patient's head (Fig. 3), there may be insufficient space for a standard tracheal tube and catheter mount to be used. All catheter mounts and connectors should be plastic to avoid the production of artifacts on images. The use of the laryngeal mask has been reported but this requires further evaluation [29].

Miscellaneous equipment

Once the patient is in the magnet bore there is no movement of any part of the magnet system, but the patient may be disturbed by the banging noise produced by gradient operation during the scan. Magnet compatible headphones or ear plugs are available, and some form of ear protection may be advisable for the unconscious patient since there have been reports of hearing loss [30]. Patient comfort is important. We have adapted a 'Spenco' mattress (Spenco-Medical, Haywards Heath, Sussex) to minimise the possibility of pressure sores developing in critically ill patients during prolonged examinations.

Needles and intravenous cannulae. Most are made of high grade stainless steel which is not attracted by the magnetic

Table 2. Anaesthesia and ventilation in a magnetic resonance unit which is without modified ventilatory and monitoring equipment.

1. Do not administer anaesthesia until you are satisfied that there are no patient contraindications to an MR examination (Table 2, Part I of this paper [1]).
2. Ensure that you are familiar with the MR installation, including the extent of fringe fields and RF shielding (Table 2, Part I of this paper [1]).
3. Ensure that the equipment needed for the technique chosen (such as the one below) is available. You may need time to modify existing equipment (see 7 below).
4. Position the anaesthetic machine well outside the 50 Gauss line.
5. Induce anaesthesia and intubate the patient's trachea outside the magnetic field.
6. Allow the patient to breathe spontaneously or ventilate by hand with oxygen and air, or oxygen and nitrous oxide using a modified T-piece system.
7. Maintain anaesthesia with either a volatile agent or with intermittent drug doses or an infusion. Infusion pump must be sited outside the 30 Gauss line and extension tubing is required. If possible, avoid moving metallic equipment once the scan has begun.
8. Monitor with an oesophageal stethoscope, a finger on the pulse if possible (the dorsalis pedis is usually accessible), a clearly visible marker placed on the chest to observe respiratory movement, and, if a second trained person is available, a capnograph with extended tubing sited on the anaesthetic machine. Try to acquire extended tubing and plastic connectors for an automated BP monitor.
9. Remove the patient from the magnet bore before reversing neuromuscular blockers or discontinuing anaesthesia.

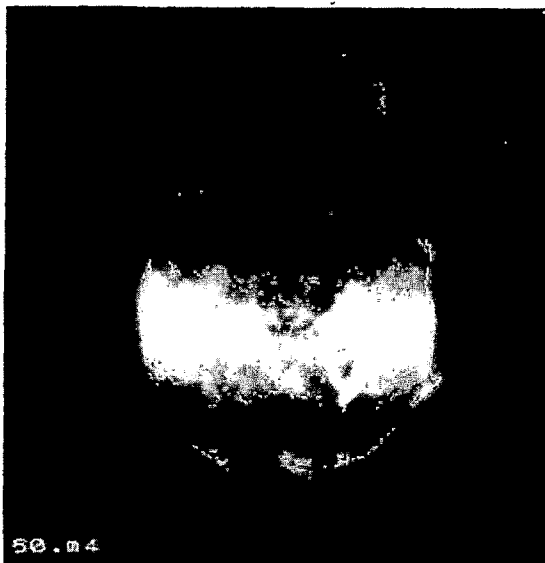


Fig. 4. Transverse magnetic resonance image of the head of a spontaneously breathing, intubated patient, who is using accessory muscles for ventilation. Artifacts result from head movement secondary to respiratory effort.

field. However, any new items of unknown content should be carefully tested for magnetic attraction before use.

Infusion pumps. The motor of an infusion pump may be damaged by the high magnetic field and result in pump malfunction. Karlik *et al.* tested an IVAC 530 pump and found this to be accurate to within 1% when sited 2 m from the face of a 1.5 T magnet [20]. We have used IVAC 710/711 and IMED 960/960A infusion pumps, in a magnetic field of approximately 30 Gauss, and found them to function accurately. The use of Vygon Lectro-Spiral (Vygon (UK) Ltd, Cirencester) extension sets eliminates the problems of trailing lines but permits them to be stretched.

Anaesthetic techniques

Anaesthesia can be maintained by a total intravenous technique [5,15,17], and we have had experience with propofol infusions. A volatile agent may also be used [15] although there is a possibility that general anaesthesia itself may alter the MRI signal from the brain [31]. The output of Cyprane TEC III and Fluotec-4 vaporizers have been tested and appear to be unaffected by the magnetic field [20,25]. The choice of spontaneous or controlled ventilation can be made by the individual anaesthetist. It is important to incorporate some form of disconnection alarm (airway pressure and end-tidal CO_2), since there is always a danger of distant disconnection.

In patients with ventilatory problems, neuromuscular blockade and controlled ventilation is preferable, not only for the benefit of improved gas exchange. Excessive neck and head movements associated with obstructed or inadequate respiration may render the imaging examination useless (Fig. 4).

The need for sedative premedication should be reviewed on an individual basis. Many seriously ill patients may be referred to the unit as day cases and may have raised intracranial pressure. Removal of secretions will not be possible when the patient is in the magnet bore. The use of glycopyrronium should be considered since it is a more

effective antisialogogue than atropine, has a longer duration of action and produces less tachycardia.

Sedation

Sedation is used in many centres to facilitate examination in children. Keeter *et al.*, in a review of current practices of paediatric sedation for computerised tomography (CT) in the USA, suggest that more than 80% of toddlers are sedated for CT [32]. An anaesthetist was involved in less than 1% of cases and in 47% of responding hospitals the sedation was prescribed by the radiologist. Commonly, the sole form of monitoring of both lightly and deeply sedated children was visual inspection from the control room and in only 11% of hospitals was any type of electromechanical monitoring device used. Current practice therefore appears to be far from ideal and has been the subject of two recent editorials [33,34]. It is probable that an even higher percentage of children will be sedated for MRI, since the examination time is longer than for CT. For reasons already stated, monitoring during MRI is difficult. Resuscitation equipment and an oxygen supply should be at hand, but for safety reasons may be inaccessible, so that when required, oxygen tubing and leads may be of insufficient length.

In this department, only two critical incidents have been recorded, out of a total of 600 or more infants and small children who have received sedation for MRI or MRS. It is usual practice for the sedation to be prescribed by the referring paediatrician, using oral chloral hydrate in a dose ranging from 30–100 mg.kg^{-1} [35,36]. Although this dose is in excess of that recommended by the *British National Formulary* (20–30 mg.kg^{-1}) it does produce a predictable level of sedation, mostly without complications. This is an important consideration in a busy radiological unit. The institution of adequate monitoring has generally confirmed, and improved, the safety of this regimen. Temazepam syrup may provide equally satisfactory levels of sedation.

In this department, neonates are not sedated, but are fed before the examination, wrapped well and placed prone within the magnet. The majority settle quickly and sleep through the study. Infants are also placed prone or on their side. Older children and adults may be examined in the supine position, but if there is concern over the airway the possibility of lateral positioning should be discussed with the radiologist. Radiologists should consult an anaesthetist about problem patients and there may be situations in which anaesthesia may be considered the safest option. Initially, examinations in this department were performed with an apnoea alarm only. Pulse oximetry and ECG is now applied by the radiographers and more than 100 infants have been monitored (with the use of radiofrequency filters) without any deterioration in the quality of images produced. The case for giving supplementary oxygen to sedated infants has been convincingly argued [34].

Cardiopulmonary resuscitation

It is impossible to manage a cardiopulmonary arrest in the bore of a magnet, therefore the patient must immediately be removed from the bore and out of the magnetic field. It is essential that staff in an MR unit practice a cardiac arrest

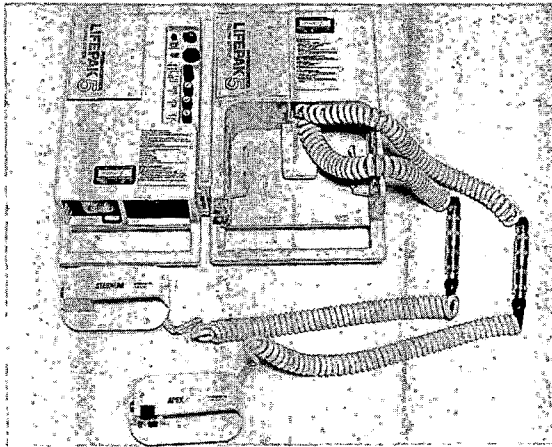


Fig. 5. Defibrillator, (Lifepak 5, Physio Control International) with leads to the paddle modified by the manufacturer, to double their length to 4 m.

drill and that each member is assigned to a particular rôle. If the hospital cardiac arrest team is called, all ferromagnetic items must be removed from them before they are allowed into the magnet suite.

Defibrillators

The unit used in this department (Lifepak 5, Physio-Control International, Basingstoke) is sited just outside the 5 Gauss line, since the batteries are ferromagnetic. The leads to the paddles have been lengthened to 4 m by the manufacturer and this has not decreased the power output (Fig. 5). The patient can be defibrillated within the magnetic field, but the defibrillator is kept at a safe distance from the magnet bore. There has been one report of defibrillator failure, when the defibrillator was kept at the edge of the magnetic field [37]. Regular testing has not revealed any reduction in its reliability or power output in the range of 20–360 J.

The cathode ray oscilloscope ECG trace on the defibrillator is distorted by the magnetic field but, in our experience, still provides adequate clinical data.

Laryngoscopes

Standard Macintosh laryngoscopes are metallic not ferromagnetic and therefore in high magnetic fields they undergo a degree of torque. The batteries, however, are highly magnetic. It has been suggested that a DC light source be used to solve this problem [20], but a simpler solution is to use a plastic Penlon laryngoscope (adult and paediatric) with a paper covered 3 V lithium battery. A nonmagnetic aluminium spacer is necessary to hold the single battery in place (Fig. 6). This adaptation is completely nonmagnetic and can be used even at the centre of the magnetic field.

Resuscitators

The Laerdal (Laerdal Medical Ltd, Orpington, Kent) adult and paediatric self-inflating silicone bags are valveless and have no magnetic working parts, therefore they can be safely used within a magnetic field.

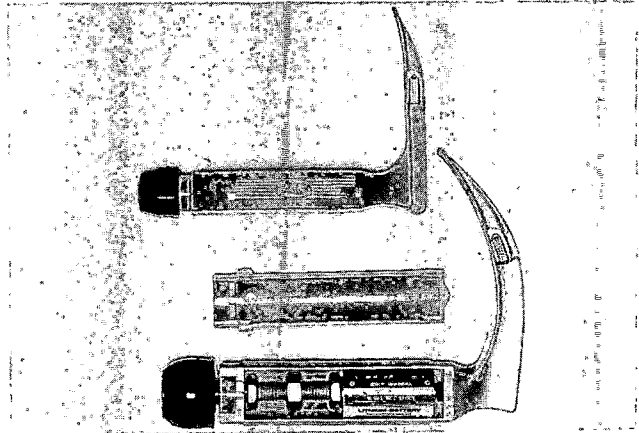


Fig. 6. Plastic laryngoscopes (Penlon) powered by a single paper covered, nonmagnetic 3 V lithium battery. A nonmagnetic aluminium spacer is required to hold the single battery in place.

Suction apparatus

A hand-held emergency suction apparatus (Vitalograph Ltd, Maids Moreton House, Buckingham, UK) is non-ferromagnetic. The wall-mounted suction unit (Ohmeda) with 10 m extension tubing can also be used.

Monitoring

Reliable and accurate monitoring is an essential component of the safe examination of an unconscious individual in an MR system, since the patient cannot be seen adequately and is not readily accessible to the anaesthetist.

The use of monitoring equipment is hindered by the problems already outlined and solutions to some of these have been discussed in previous papers [38,39]. Many manufacturers of MR equipment now make ECG and respiratory monitors compatible with their own systems, which are primarily used for respiratory and cardiac gating of imaging sequences but may in addition be used for monitoring. Although many of these cannot be relied on to provide any qualitative assessment of the ECG (manufacturers' product information will specifically disclaim any diagnostic attributes), they can still be used to provide an index of heart rate and serve as a gating signal for other monitoring equipment (e.g. pulse oximeters: see below). The use of fiberoptic [40] and telemetry systems [41] avoids the hazards of trailing cables.

Burns. Recent reports of burns resulting from current induction by the oscillating radiofrequency fields in monitoring leads are of concern [42,43], and adherence to the safe practice guidelines suggested in Part I is important [1].

ECG

The ECG of an individual situated within a static magnetic field shows significant changes [44]. According to Faraday's law, a voltage is induced across a column of conducting fluid (in this case blood) flowing through a magnetic field. The changes are maximal when the fluid flow is at 90° to the field, which occurs in a supine patient when blood flows through the transverse aorta, hence the changes are principally in leads I, II, V_1 and V_2 [45]. These superimposed potentials are greatest in the early T waves, and late ST

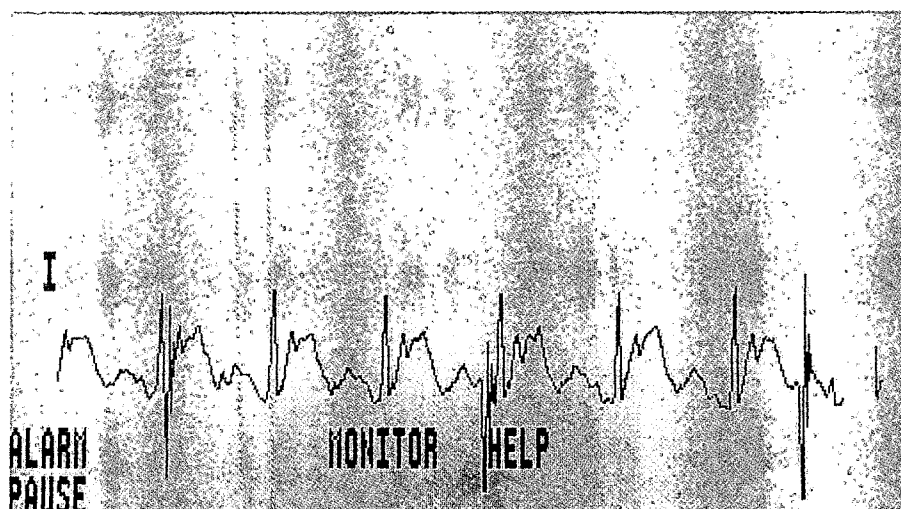


Fig. 7. ECG recorded from a healthy volunteer within the bore of a 1.6 T magnet. The T wave and ST abnormalities, caused by the voltage induced by blood flow, are clearly shown. The changing magnetic fields of the imaging gradients have produced spike artifacts superimposed on the ECG.

segments and can mimic the ECG changes of conditions such as hyperkalaemia and pericarditis (Fig. 7). They are related to field strength and hence increase as this increases.

The rapidly changing magnetic fields of the imaging gradients also induce potential differences across the loops formed by the ECG leads. Artifacts produced by the pulsing of the gradients can appear as spikes on the ECG and can be of sufficient magnitude to be detected and interpreted as an R wave. They cause interference with cardiac gating and an artificially elevated heart rate on the ECG monitor (Fig. 7).

The radiofrequency currents used to excite the nuclei during MRI can cause induction of currents within leads and cables and produce artifacts on the ECG. This problem can be ameliorated by the use of shielded cables [1]. Another solution is to transmit the ECG data by telemetry [41], lossy transmission lines [46] or a fiberoptic system [40].

The use of electrodes or leads which contain metal will exacerbate the problem of current induction in addition to producing image artifacts [47]. We have successfully used carbon fibre leads (NDM Division, American Hospital Supply Corporation, Dayton, Ohio, USA), similar to those described by van Genderingen *et al.* [48]. Some ECG electrodes are magnetic, but 'Red Dot' (3M Medical, St. Paul, Minnesota) and Medicotest (Medicotest A/S, Olstykke, Denmark) do not appear to cause interference.

Careful placement of the ECG electrodes and leads can minimise the ECG changes produced by the static and changing magnetic fields. The recommendations of Wendt *et al.* [47] and Dimick *et al.* [45] are summarised here: (1) braid or twist the leads to reduce the loops across which potential differences can be generated; (2) place the ECG electrodes as close as possible to the centre of the magnetic field since the gradients should be changing least at this point; (3) if possible keep the limb electrodes close together and in the same plane; (4) when using the chest leads, V_5 and V_6 are least likely to develop and record artifacts because they are close to the QRS axis, and far from the voltage induced by blood flow through the transverse aorta.

Two other points are worth noting. The relative length of ECG leads may be critical in preventing gradient-induced artifact, therefore many manufacturers advise against modifying ECG leads supplied with MR systems. If a cathode ray tube ECG monitor is used the magnetic field will pull the beam of electrons off centre and distort the trace. A liquid crystal display screen, such as that used in the Marquette (Marquette Electronics Inc., Bewdly, Worcs.) 'TRAM' system, does not undergo distortion and provides a clear backlit display.

Pulse plethysmography [49] and standard vascular Doppler systems [39] have also been used to show heart rate and assess peripheral perfusion.

Pulse oximetry

Pulse oximetry has proved to be the most difficult monitor to use within magnetic fields. The problems and possible solutions are summarised in Table 3.

We originally tested four pulse oximeters in our 1.6 T magnet; the Nellcor N-100, the Nellcor N-200, the Datex OSP-200 and the Ohmeda Biox 3700. The Nellcor N-200 consistently gave the best performance, and we have now used it on approximately 100 patients, including preterm neonates and adults with poor peripheral perfusion.

There are several reasons that may account for the Nellcor N-200's performance in the MR environment. All the pulse oximeter leads were passed through radiofrequency filters. The Nellcor is the only oximeter in which the signal is amplified by a nonmagnetic pre-amplifier before it is passed through the filters, therefore there is less loss of signal during filtering. It also has an ECG locking facility which improves detection of the pulse waveform when interference is present (Fig. 8). In addition, the standard Nellcor probes have no magnetic components, therefore they do not require modification.

McArdle *et al.* used Nellcor N-100 and Ohmeda Biox pulse oximeters for neonates [36]. They found slight image degradation with the Nellcor and significant loss of quality with the Ohmeda Biox. We noted no change in spectral or image quality when the leads were passed through the

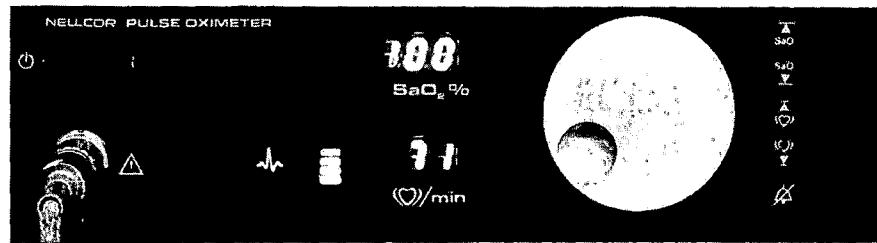


Fig. 8. Nellcor N-200 pulse oximeter in use during a magnetic resonance imaging examination. The ECG lock facility (QRS complex symbol) is in use, helping to detect the pulse waveform when interference is present.

radiofrequency filters. Aluminium foil has been used to increase the shielding of the pulse oximeter probe [50]. We originally used variable length plastic radiofrequency shielding (Alpha Wire Corporation, USA) to enclose leads. However, we found the use of radiofrequency filters alone to be satisfactory, provided that an ECG signal was available to gate out gradient artifact.

Karlik *et al.* tested Nellcor, SensorMedics and Ohmeda Biox pulse oximeters and, in contrast to our experience, found the Ohmeda Biox 3700 to give the best results. They noted radiofrequency interference even with the oximeter cable shielded and suggested using an optical link for the infrared signal [20].

The Nonin 8604 (Cardiokinetics, Salford) is a new MR-compatible pulse oximeter. After limited testing we found it performed well. It provided continuous data during imaging and there was no image degradation. This unit is now available with a fibreoptic sensor (8604FO) which should further improve data quality and reduce the risk of radiofrequency-induced burns.

Capnography

We have successfully used an Ohmeda 5200 capnograph sited at a distance of 11 m from the patient's head, although the lag and alarm times are about 10 s. The waveform shows a prolonged upslope (Fig. 9), even in individuals with healthy lungs, because of the long gas pathway. Although end-tidal concentrations of CO₂ are not accurate, trends can be observed and the respiratory rate estimated.

A nasal catheter has been used to monitor end-tidal carbon dioxide in spontaneously breathing patients [51]. We have found this technique useful in sedated infants. A

14-gauge cannula inserted into one limb of nasal oxygen prongs has also been used to monitor expired CO₂, in sedated patients [52]. The use of a commercially available capnograph (8800 Capnometer, Biochem International) and respiration monitor (515 Respiration monitor, Biochem International) has also been described [53].

Noninvasive blood pressure

Satisfactory NIBP readings are obtained if the ferrous connections on a standard automated (Dinamap) cuff are changed to nylon and the tubing is lengthened as necessary.

Invasive pressure monitoring

Direct pressure readings can be obtained if the lead from the pressure transducer is passed through a radiofrequency filter. The transducers must be kept close to the patient (within 1.5 m) so that damping caused by excessively long saline filled columns, is prevented [19]. The waveforms are displayed on our Marquette 'TRAM' monitor liquid crystal display, without any loss of accuracy of reading or damping of the trace. This is to be expected since the radiofrequency filters are filtering from the kilohertz range upwards, while the clinical waveforms have harmonics below 100 Hz. Intracranial pressure monitoring has been described using nonmetallic subarachnoid bolts [19].

Precordial/oesophageal stethoscopes

These may be unsatisfactory because of the length of tubing required and the fact that the noise made by the gradients changing during the examination may obscure the heart and breath sounds.

Table 3. Problems encountered in the use of pulse oximetry with magnetic resonance systems and some possible solutions.

1. Degradation of pulse oximetry signal by MR	
Problem	Solution
Poor signal due to movement	Careful selection of site and placement of probe.
Poor perfusion or contact	
Induced currents in cable	Screen cable.
	Minimise cable length.
Poor signal/noise ratio	Amplify signal early.
2. Degradation of MRI by pulse oximeter	
Problem	Solution
Metallic artifact on images	Remove all magnetic components from probe or use plastic/disposable probes.
Interference due to switched pulses	Screen cable.
Introduction of stray radiofrequency current by leads acting as aerials	Screen lead. Pass through radiofrequency filters, or keep unit within radiofrequency screened room.

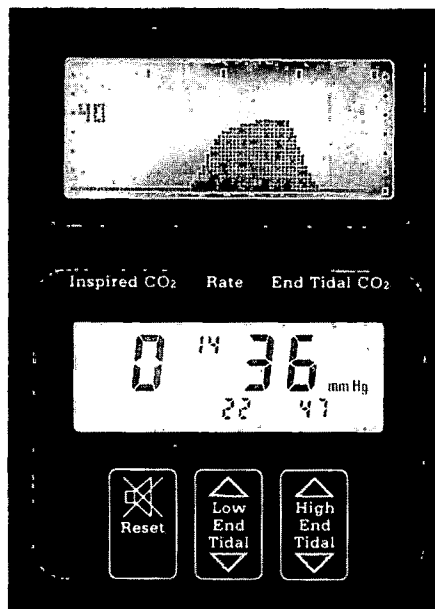


Fig. 9. Expired CO₂ trace of a normal volunteer with no respiratory disease, using 11 m long tubing. The prolonged upslope, resulting from the long gas pathway, is shown.

A wireless stethoscope, using infrared transmission with a range of 10 m for distant monitoring has been designed by Hök *et al.* [54]. The system has been modified to suppress MR generated noises, using specially developed precordial or nasal pickup heads connected to polymer tubing. Experimental tests within an MR system have shown that common mode acoustic noise is suppressed by about 30 dB in the relevant frequency region, and initial clinical use has proved satisfactory [55].

Temperature

We have not encountered problems in monitoring peripheral and central sites with temperature probes (Marquette Systems Inc.) using radiofrequency filters.

Integrated monitoring units designed to work in high magnetic fields are being developed. One such unit, recently available, the MAGLIFE (Adam, Bruker Group, Wissembourg, France) displays and monitors ECG, respiratory rate, capnography, oxygen saturation, invasive and noninvasive blood pressure and temperature.

Patient transport

Standard hospital trolleys (and wheel chairs) are ferromagnetic, and so great care should be taken with them near the magnet. We have designed a mobile unit for critically ill patients which consists of a high quality stainless steel trolley (Thackray Surgery Ltd, Leeds), on which our adapted monitoring and resuscitation equipment is carried. Thus a critically ill patient is moved only once. All MR compatible monitoring leads are attached in the ICU, so that the patient can be slid from the trolley, which locks on to the patient couch, directly into the magnet bore. It is essential, however, that the ferrous oxygen cylinder is removed from the trolley well outside the 50 Gauss line.

Forward planning

Ideally the anaesthetist should be involved at the planning stage of an MR unit; unfortunately this often does not happen. The installation of piped medical gases avoids the need to change potentially dangerous ferromagnetic cylinders during an examination. Provision of an area away from the magnetic field for quiet induction, recovery and resuscitation improves both patient safety and throughput of work. The introduction of appropriate radiofrequency filters in the magnet shielding at the time of magnet installation, in consultation with the manufacturing company or the hospital medical physics department, and installation of isolated filtered alternating current power circuits, permits effective monitoring from the outset. With the provision of filtered monitoring equipment, good safety procedures and adequate space to work in, anaesthesia for magnetic resonance studies can be straightforward, and the information gained can be of an extremely high quality.

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Forum

Pre-registration house surgeons

A questionnaire study of anaesthesia-related knowledge and approach to pre-operative investigations

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Summary

A group of newly qualified preregistration House Officers completed a questionnaire relating to their knowledge of anaesthetic drugs and to their appreciation of complications which may, in whole or in part, have required some knowledge of anaesthesia. Considerable gaps in knowledge were demonstrable, not only in matters that might arguably be regarded as strictly within the province of anaesthesia, but also in respect of basic pharmacology. The same House Officers were also questioned as to the necessity for various basic pre-operative investigations prior to six everyday surgical procedures. This demonstrated a marked propensity for House Officers to overinvestigate patients as compared to the requirements of practising anaesthetists. The discrepancy was most marked with respect to pre-operative chest X rays. However, considerable disparity was also demonstrable amongst a group of experienced anaesthetists as to their requirements for pre-operative investigations. Permitting students greater exposure to anaesthesia in the undergraduate curriculum could go a long way towards improving this situation.

Key words

Investigations; pre-operative.

Training; anaesthesia, undergraduate.

Seven years ago a survey [1] revealed a number of potentially serious gaps in the knowledge of surgical house staff relating to anaesthetic drugs and practices. If this is still the case, the ability of House Officers to diagnose and manage certain problems arising in the postoperative period could be impaired and such problems may well be encountered from the first day of their appointment. In recent years the place and extent of anaesthetic input into the undergraduate curriculum has been the subject of much attention, including the publication of a discussion document by the College of Anaesthetists [2] and an editorial in the *British Journal of Anaesthesia* [3].

The aims of this study and questionnaire were chiefly twofold. Firstly, to see if the situation with regard to anaesthesia-related knowledge possessed by preregistration House Officers had changed and secondly to ascertain their approach to pre-operative investigations prior to surgery and anaesthesia. This questionnaire differed from the previous one [1] in several respects. Firstly, the list of drugs was different, reflecting evolving clinical practice. For example a question was included relating to the recognition of respiratory depression as a complication of epidural opioids now increasingly used as a means of providing a superior quality of postoperative analgesia in both teaching and district general hospitals. Secondly, a series of questions were included relating to pre-operative preparation and the ordering of investigations prior to elective surgery.

Thirdly, a more homogeneous group of doctors were studied in that they were all preregistration House Officers and they completed the questionnaire during their induction course on the first day of their appointment. Thus their knowledge and attitudes would be a function of what they had learnt during their undergraduate training and would not be influenced by any clinical experience as House Officers.

A preliminary study was undertaken in August 1989 with the new intake of House Officers at the Royal United Hospital, Bath. The results of this preliminary study suggested that a number of drugs utilised outside, as well as within, anaesthetic practice were the subject of a surprising degree of misconception. Indeed only a minority could correctly identify drugs such as droperidol, doxapram, neostigmine, edrophonium and ephedrine. This questionnaire also suggested that up to 30% of the House Officers might not readily appreciate pneumothorax as a complication of central venous cannula insertion via the subclavian route, and only a minority recognised respiratory depression as a complication of epidural opioids (Table 1). With regard to pre-operative investigations, a significant minority (22%) still felt that any patient undergoing general anaesthesia required a pre-operative estimation of urea and electrolytes whilst the majority (61%) would routinely order a chest X ray prior to surgery and anaesthesia in any patient over 60 years old.

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Table 1. Results from preliminary questionnaire (Royal United Hospital, Bath 1989) relating to anaesthetic procedures, complications and pre-operative preparation.
% indicates the percentage answering appropriately.

Question		
1.	Why is cricoid pressure applied during the induction of anaesthesia for emergency surgery?	78%
2.	What would you suspect if a patient complains of cough and dyspnoea after the insertion of a central venous line via the subclavian route?	67%
3.	Following tracheal intubation at a cardiac arrest you can only hear breath sounds over the right lung field. What would you suspect and what would you do?	94%
4.	What is the likely cause of a patient fitting 30 min after the insertion of a brachial plexus block using lignocaine via the axillary approach?	44%
5.	What is the major risk of providing postoperative analgesia using an epidural morphine (or other opioid) infusion?	28%
6.	What is the likely cause of diplopia and a weak cough shortly after major abdominal surgery?	11%
7.	A patient with a thoracic epidural receives a bolus of bupivacaine and shortly after the blood pressure decreases to 80/50 mmHg. What is your immediate treatment?	72%
8.	A young, fit patient rapidly develops a temperature of 41°C after a short general anaesthetic. What possible cause must be considered?	83%
9.	Why is the presence of a hiatus hernia relevant to the anaesthetist prior to general anaesthesia?	78%
10.	All patients undergoing surgery with general anaesthesia require pre-operative estimation of urea and electrolytes.	78%
11.	All patients for general anaesthesia over the age of 60 years require a pre-operative chest X ray.	39%
12.	Antihypertensive drugs should be withheld on the morning of operation.	83%
13.	Anticoagulant therapy is a contraindication to spinal and epidural anaesthesia.	56%

Methods

The study questionnaire was altered prior to the 1990 intake of House Officers aiming to reduce ambiguities and to minimise misinterpretation of the responses. Instead of being asked to 'indicate the nature of a drug by means of a word or phrase', respondents were asked to tick the appropriate selection from a number of choices. With regard to pre-operative investigations, six typical surgical patients were identified and respondents were asked to indicate precisely which investigations they would order in each particular case. At about the same time a group of anaesthetists were asked to indicate their requirements in respect of investigations necessary before surgery for the same six surgical patients.

Results

The questionnaire was completed by 23 out of an intake of 26 preregistration House Officers during the course of their morning induction programme on the first day of their appointment at Southampton General Hospital. All 23 completed the 'investigations' section whilst 22 completed the drugs and complications section.

'Nature of drugs' questions. The mean correct score for the questions was 15.4 out of a possible 25 with a range of 6–23. Table 2 shows the number of House Officers correctly identifying each drug and the number making no attempt at a response; some of the misconceptions are shown on the right of the table. It is interesting to note that it is not only drugs confined to anaesthetic practice that are unrecognised, but that there exists a degree of ignorance about drugs in widespread use e.g. papaveretum (although we suspect 'omnopon' would have produced 100% correct response rate!) as well as 'standard' drugs from basic pharmacology such as edrophonium and ephedrine.

'Complications' questions (Table 3). The potential for pneumothorax following subclavian venous line insertion was recognised by 21/22 whilst 19/22 correctly identified intubation of the right main bronchus. In question 3, 15/22 related the generalised muscle pains to the muscle relaxant,

Table 2. Drug recognition: number (%) of House Officers responding correctly; number not attempting a response and some misconceptions (total of 22 House Officers).

Drug	Correct response (%)	No response	Misconceptions
Propofol	5 (23)	12	Inhalational anaesthetic, anticholinergic, benzodiazepine
Isoflurane	21 (95)	—	Intravenous anaesthetic
Droperidol	7 (32)	13	Anticholinergic, muscle relaxant
Lignocaine	20 (91)	1	
Haemaccel	22 (100)		
Vecuronium	13 (59)	8	Intravenous anaesthetic
Etomidate	2 (9)	18	Local analgesic
Thiopentone	19 (86)		Inhalational anaesthetic, muscle relaxant, benzodiazepine
Bupivacaine	11 (50)	9	Intravenous anaesthetic
Ketamine	7 (32)	13	
Glycopyrronium	3 (14)	18	
Hyoscine	20 (91)	1	
Ephedrine	15 (68)	5	Anticholinergic, anticholinesterase
Naloxone	15 (68)	5	Sympathomimetic
Hetastarch	7 (32)	12	
Midazolam	17 (77)	4	
Enflurane	14 (64)	7	Intravenous anaesthetic
Neostigmine	19 (86)	2	Muscle relaxant
Suxamethonium	19 (86)	2	Anticholinergic
Alcuronium	11 (50)	10	Anticholinergic
Nitrous oxide	22 (100)		
20% albumin	15 (68)	6	
Papaveretum	7 (32)	11	Muscle relaxant, sympathomimetic, intravenous anaesthetic, local analgesic
Edrophonium	15 (68)	5	Muscle relaxant, anticholinergic
Doxapram	11 (50)	4	Benzodiazepine, sympathomimetic

Table 3. The 'complications' part of the questionnaire and the numbers giving the correct response.

In a word or a phrase indicate the most likely cause of the complication or problem described.	
1. What would you expect if a patient complains of cough and dyspnoea after the insertion of a central venous line via the subclavian route?	21/22
2. Following tracheal intubation at a cardiac arrest you can only hear breath sounds over the right lung field. What do you suspect?	19/22
3. Four fit, young patients undergo wisdom teeth extraction one afternoon and receive the same anaesthetic technique. The following day two of the patients complain of generalised muscle pains and stiffness. What is the probable cause?	15/22
True/False (circle the correct answer)	
1. Regular antihypertensive medication should be withheld on the morning of operation.	True/False 19/22
2. Regular oral hypoglycaemic medication should be withheld on the morning of operation.	True/False 17/22
3. Infants under one year must be starved for at least 6 h prior to surgery and anaesthesia.	True/False 3/22
4. Anticoagulant therapy is a contraindication to spinal and epidural anaesthesia.	True/False 13/22

but not all specifically implicated suxamethonium, whilst others confused the aetiology with pseudocholinesterase deficiency.

However, it was encouraging that 19/22 appreciated that antihypertensive medication should be continued up to the time of operation whilst 17/22 recognised the need to omit oral hypoglycaemic drugs on the morning of operation. However, 19/22 wrongly believed that infants under one year of age should be starved for at least 6 h prior to surgery and anaesthesia whilst only slightly over half (13/22) recognised anticoagulant therapy as a contraindication to spinal and epidural anaesthesia.

'Pre-operative Investigation' questions. Table 4 shows the number and percentage of House Officers who would order a particular investigation for a particular patient. However, in order to make these results more meaningful they should be set against what a practising body of anaesthetists would require in the preparation of that patient. The same questionnaire was given to 32 anaesthetists

(14 Consultants, eight Senior Registrars, five Registrars and five Senior House Officers) asking them which investigations they would require.

Figure 1 illustrates the variation in investigations required with respect to the grade of anaesthetist and demonstrates some areas where the profession would appear to be divided on the necessity of a particular test. Moreover, this is not solely explicable in terms of the more experienced anaesthetist requiring fewer tests, although there is a trend in that direction.

Figure 2 shows the number of House Officers ordering a test, set against the number of Consultants and Senior Registrars actually requiring it. The wide difference in many of the columns indicates the tendency for House Officers to overinvestigate. In no investigation for any operation did the propensity of the House Officer to investigate fall below the requirements of a Senior Registrar or Consultant.

Discussion

The place and value of anaesthesia in the undergraduate curriculum has been the subject of some attention. Whilst the detail and practice of clinical anaesthesia is appropriately a matter for specialist postgraduate training, attachments to departments of anaesthesia have a lot to offer students in terms of revising and placing in a clinical context much of the physiology and pharmacology learnt at earlier stages in the medical course. In addition, a valuable opportunity is provided to practise and gain expertise in everyday practical procedures such as venous cannulation and tracheal intubation.

Many medical schools give anaesthesia a low priority when allocating time in the undergraduate curriculum and so lose the opportunity of integrating much of the basic science teaching to the management of surgical patients. The recent discussion document from the College of Anaesthetists [2] recommends that a minimum of 2 weeks (and preferably longer) be devoted to a course of instruction run by a department of anaesthesia. However, the document stresses the necessity that such courses would require adequate funding and proper recognition of the manpower implications if there is to be no curtailment of ongoing clinical and research commitments.

One of the major roles of the newly qualified surgical House Officer is in the pre-operative preparation of

Table 4. Number (%) of House Surgeons requesting each test, prior to surgery and anaesthesia (total of 23 housemen).

	Hb	U&E	ECG	CXR
1. Fit 40-year-old requiring hysterectomy for menorrhagia	23 (100)	17 (74)	0 (0)	1 (4)
2. Fit 65-year-old for an inguinal hernia repair	21 (91)	18 (78)	20 (87)	17 (74)
3. Stable but restricted 70-year-old chronic bronchitic (ECG and CXR available from 6 months previously), for a check cystoscopy	22 (96)	17 (74)	14 (61)	15 (65)
4. Healthy 7-year-old for an orchidopexy	3 (13)	2 (9)	0 (0)	1 (4)
5. Fit 35-year-old for a breast biopsy but with signs of reduced air entry to right chest	16 (70)	8 (35)	2 (9)	23 (100)
6. Well controlled 55-year-old hypertensive receiving a thiazide diuretic for total hip replacement	23 (100)	23 (100)	22 (96)	18 (78)

Hb, haemoglobin estimation;
U&E, urea and electrolyte estimation;
ECG, electrocardiograph;
CXR, chest X ray.

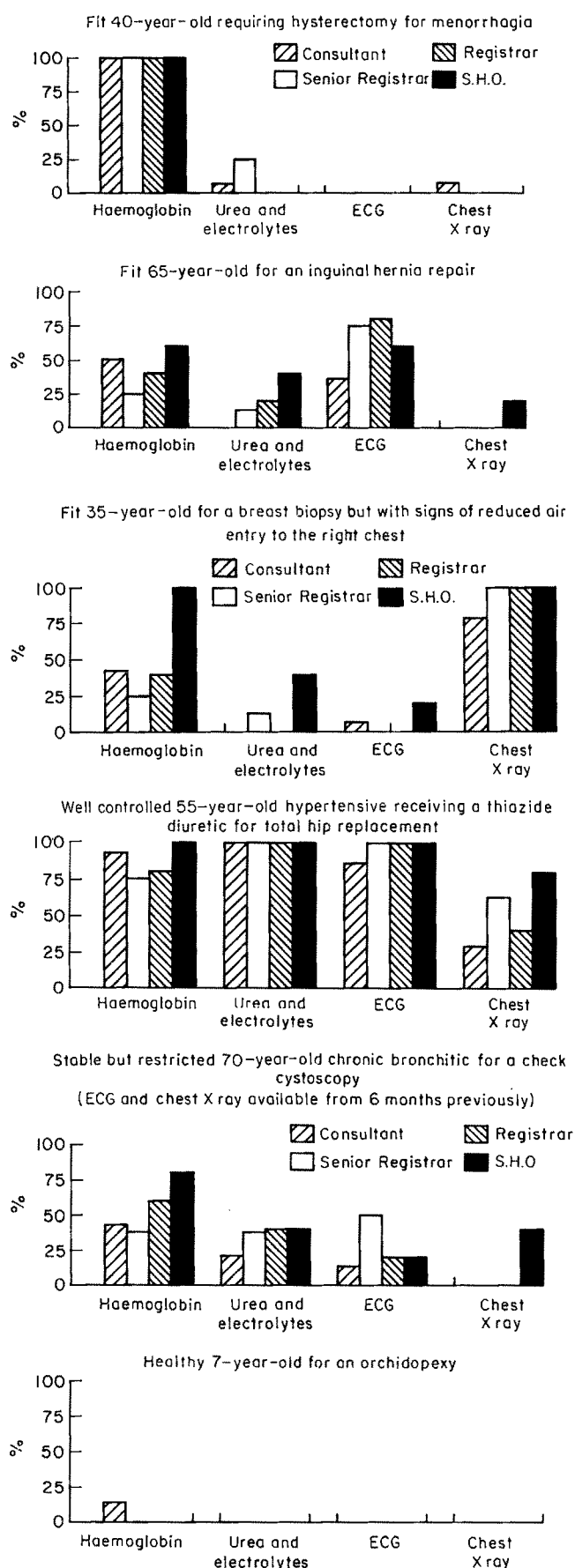


Fig. 1. Relationship between grade of anaesthetist and tendency to require a particular pre-operative investigation. Of the 32 respondents 14 were Consultants, eight Senior Registrars, five Registrars and five Senior House Officers.

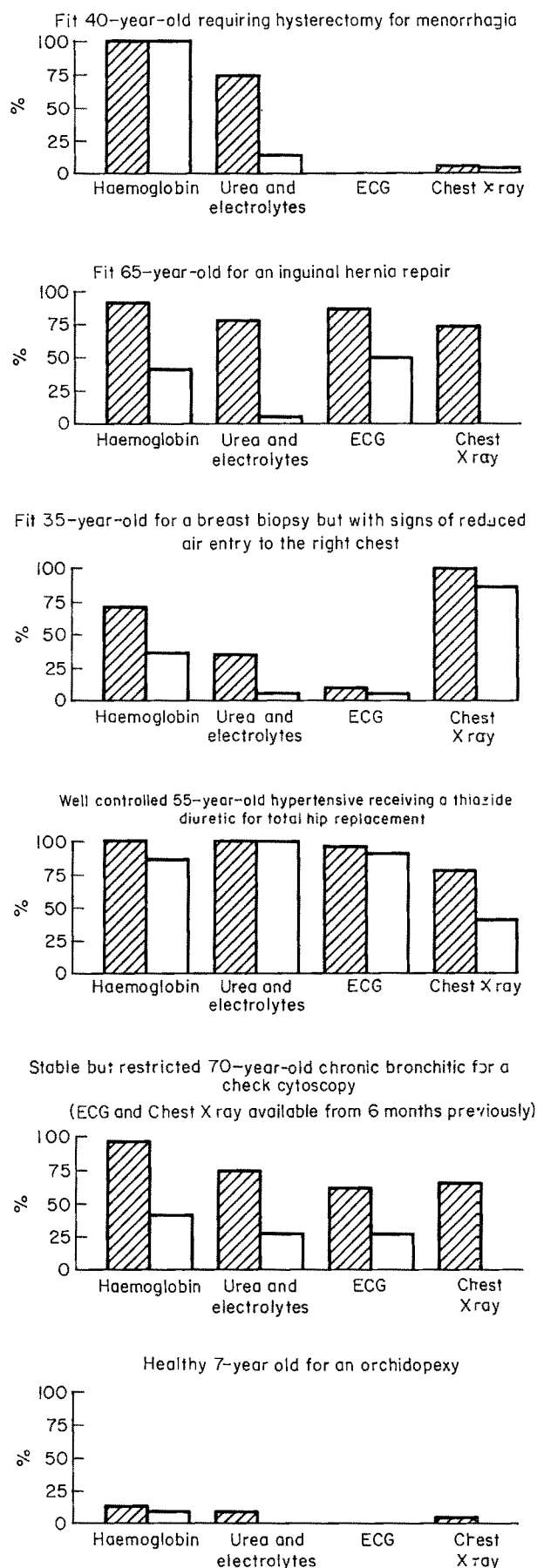


Fig. 2. Percent of House Surgeons ordering a test set against percent of Senior Registrar and Consultant Anaesthetists requiring it. House Surgeons (▨) $n = 23$; anaesthetists (□) $n = 22$.

patients for surgery and the subsequent management of problems arising in the postoperative period. Once a patient has been returned to the ward after surgery, the House Officer may be called upon to attend to potentially life-threatening problems whose aetiologies may in part lie in the technique of anaesthesia and whose diagnosis will be facilitated by a knowledge of anaesthetic drugs and techniques. The results of this questionnaire leave little doubt that considerable gaps in relevant knowledge still exist.

Similarly, the pre-operative preparation and investigation of patients prior to elective surgery is frequently poorly controlled and 'protocols', where they do exist, frequently have their origins in word of mouth handed down from one generation of House Officers to another. Omitted investigations may lead to patient cancellation with the attendant waste of valuable theatre time. However, the findings of this study suggest that errors of commission are far more common than errors of omission with patients being needlessly overinvestigated; perhaps for fear of incurring the wrath of anaesthetic colleagues! Such high levels of inappropriate investigation across the country must inevitably result in a subtle but not inconsiderable cost burden to the NHS.

In this study considerable disparity between the propensity for a House Officer to order a particular investigation and it actually being required by the anaesthetist (Consultant or Senior Registrar) was demonstrable for all four of the investigations studied (Fig. 2). However, this is most strikingly so, and has the greatest cost implications, when chest X rays are considered.

Baseline pre-operative chest X rays have been advocated by a number of authors, particularly in elderly patients [4, 5]. However, there is now evidence suggesting that routine pre-operative chest X rays are of no value and should only be performed when there is a specific clinical indication [6-9]. The view of the Royal College of Radiologists is that 'routine' films can no longer be clinically justified [10]. Even studies demonstrating the detection of a high incidence of unexpected radiological abnormalities in elderly groups fail to demonstrate conclusively that the availability of a pre-operative film would facilitate the postoperative management of a complication [11].

Few would argue that the undergraduate course should provide a broad based education rather than a didactic training to perform the duties of a specific post. Yet it seems clear that there is a strong case for a greater input from anaesthetists both to the undergraduate curriculum and the preregistration year. In the case of the preregistration year, anaesthetists could specifically cover the management of postoperative complications and agree to disseminate rational protocols for pre-operative preparation and investigation. This could be achieved by allocating anaesthesia a greater role in the induction programmes for newly appointed junior doctors and this input should enable the House Officer to recognise and react to problems with greater confidence and competence.

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Isosorbide dinitrate spray

Attenuation of cardiovascular responses to laryngoscopy and intubation

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Summary

We evaluated the efficacy of isosorbide dinitrate buccal spray (Isomack) in attenuating the cardiovascular response to laryngoscopy and tracheal intubation in 60 patients undergoing elective surgery under general anaesthesia. Patients were allocated to one of three groups of 20 patients each. Group 1 patients were administered placebo buccal spray 90 s before induction of anaesthesia. Groups 2 and 3 had isosorbide dinitrate spray 30 and 90 s before induction of anaesthesia. Systolic, diastolic and mean arterial pressures and heart rate were monitored. After the spray, group 3 patients had a significant decrease in systolic arterial pressure ($p < 0.01$). At 1 min after intubation, systolic, diastolic and mean arterial pressures showed a significant increase in group 1 patients (24.9 mmHg, 14.2 mmHg and 18.7 mmHg respectively). In contrast, groups 2 and 3 showed a significant decrease in these parameters ($p < 0.01$). Although significant tachycardia was present following intubation in all the three groups, the degree of tachycardia was greater in groups 2 and 3 ($p < 0.01$).

Key words

Anaesthetic techniques; laryngoscopy, tracheal intubation.

Blood pressure; hypertension.

Pharmacology; isosorbide dinitrate spray.

Transient responses in the form of tachycardia, arterial hypertension and cardiac arrhythmias during laryngoscopy and tracheal intubation are well documented [1–3]. These responses are harmless in healthy individuals, but in those with conditions such as hypertension, coronary artery disease, cerebrovascular pathology and leaking abdominal aneurysm, serious complications can occur [4–6].

A number of drugs have been used in an attempt to attenuate these responses, but with variable success [7–18]. Isosorbide dinitrate, a high molecular weight organic nitrate, is available as oral, chewable and sublingual tablets, sustained release capsules, ointment, and more recently as a buccal spray [19]. In patients with angina, the buccal spray had a faster onset of action, earlier haemodynamic changes and a longer duration of action than the sublingual tablets [20–21].

In view of the widespread use and apparent safety of the buccal spray for patients with angina, we decided to evaluate its efficacy in attenuating the cardiovascular responses to laryngoscopy and tracheal intubation in normotensive patients. In addition, we wished to evaluate the interval between administration and peak action of the preparation.

Material and methods

Sixty ASA grade 1 patients of either sex, aged between 15 and 35 years, scheduled for routine elective surgery, participated in this blind, controlled, randomised study. Ethics committee approval was received and informed consent was obtained from all patients. Those in whom the duration of laryngoscopy and intubation was greater than 30 s were excluded.

Premedication consisted of oral diazepam 0.2 mg.kg^{-1} given the night before and again 2 h before surgery. Morphine 0.15 mg.kg^{-1} and promethazine 0.5 mg.kg^{-1} were also given intramuscularly 1 h before surgery.

On arrival in the operating theatre, an intravenous cannula was inserted and after a resting period of 2 min the following parameters were recorded: (a) effect of premedication [22]; (b) systolic (SAP), diastolic (DAP) and mean (MAP) arterial blood pressure (automatic sphygmomanometer) and heart rate (ECG monitor). Patients were allocated to one of the following three groups with the help of a randomisation chart.

The isosorbide dinitrate preparation used (Isomack, Heinrich Mack NACHF, D-7918 Illertissen, Federal Republic of Germany) is provided in a metal aerosol container. One spray delivers 1.25 mg isosorbide dinitrate in 0.9 ml.

Group 1 patients received a placebo spray 90 s before induction of anaesthesia. Group 2 patients received buccal isosorbide dinitrate (one spray) 30 s before induction of anaesthesia. Group 3 patients received buccal isosorbide dinitrate (one spray) 90 s before induction of anaesthesia.

Patients were questioned about any burning sensation in the mouth following the spray, and other side effects such as skin rash and hypotension were looked for. Anaesthesia was induced with thiopentone and tracheal intubation was facilitated with suxamethonium chloride (2 mg.kg^{-1}). Ventilation of the lungs was carried out with 33% oxygen in nitrous oxide using a Bain system.

Arterial pressures and heart rate were monitored according to the following time sequence (Fig. 1): (a) basal reading (B); (b) 1 min after the spray (B₁); (c) 1.5 min after

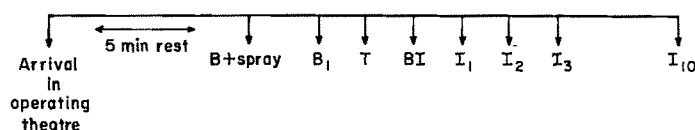


Fig. 1. Sequence of cardiovascular monitoring. B, basal; B₁, 1 min after spray in groups 1 and 3 but before suxamethonium in group 2; T, after thiopentone; BI, before intubation; I₁, 1 min after intubation; I₁₀, 10 min after intubation.

thiopentone (T); (d) before intubation (BI); (e) 1 min after intubation and then every min up to 10 min after intubation. These readings were labelled as I₁, I₂, I₃, I₄ etc. The timing of cardiovascular monitoring is shown in Figure 1. Arterial blood gas samples were taken before and after intubation to ensure normocarbida. Pancuronium bromide was given for neuromuscular blockade.

All the observations were analysed statistically using Student's paired *t*-test and analysis of variance test to compare the changes within the groups and between the groups respectively.

Results

Patients were well matched with regard to age and sex distribution (Table 1) and the pre-operative cardiovascular parameters (Table 2).

Systolic arterial pressure (SAP) (Fig. 2). Group 1 showed an increase in SAP, whereas groups 2 and 3 had a decrease in SAP at 1 min following spray administration. However, only the decrease seen in group 3 (6.6%) was

statistically significant ($p < 0.01$). Maximum changes in SAP occurred after 1 min following intubation. The mean increase in group 1 was 24.9 mmHg (19.65%) while the mean decrease was 25.6 mmHg (20%) and 26.3 mmHg (20.4%) in groups 2 and 3 respectively. These changes were significant within the groups up to 8 min. Also, whilst the difference in SAP between groups 1 and 2 and 1 and 3 was significant up to 8 min, it was significant between groups 2 and 3 only up to 2.5 min in the postintubation period.

Diastolic arterial pressure (DAP). Apart from variations in the magnitude and duration, changes in DAP were similar to those seen in the SAP. The increase in DAP in group 1 at 1 min following intubation was 14.2 mmHg (15.5%) while the decrease was 8.8 mmHg (10.01%) and 11.4 mmHg (12.9%) in groups 2 and 3 respectively. These changes persisted for 5 min during the postintubation period (Table 3).

Mean arterial pressure (MAP). The pattern of changes in MAP was similar to SAP and DAP. These changes were significant within the groups up to 7 min in the postintubation period and between groups 1 and 2 and 1 and 3 up to 10 min. However, the difference in MAP between groups 2 and 3 was significant up to 2 min only (Fig. 3).

Heart rate (HR) (Fig. 4). Significant tachycardia was present in all the three groups following both the spray and tracheal intubation. However, a significantly greater increase in HR was seen (12.3% and 18.35% in groups 2 and 3 respectively) compared with group 1 (4.31%) following the spray. The increase in HR following intubation was 17.15%, 22.19% and 27.87% in groups 1, 2 and 3 respectively and this increase was significant within the groups. On analysis between the groups, groups 2 and 3 had significantly greater tachycardia ($p < 0.05$) compared with group 1. However, the degree and duration of tachycardia was statistically comparable between groups 2 and 3 (Fig. 3). No other side effects of isosorbide dinitrate were seen following the spray administration in groups 2 and 3. Arterial blood gases taken during the study period were normal.

Discussion

Isosorbide dinitrate, which acts on almost all smooth muscle throughout the body, is a major component of anti-anginal drug regimens [19, 20, 23]. It has been used as a

Table 1. Patient data. Values are expressed as mean (SD).

	Group 1	Group 2	Group 3
Age; years	22.2 (4.24)	21.6 (3.91)	21.9 (4.33)
Weight; kg	48.3 (4.29)	47.8 (3.74)	48.3 (3.68)
Sex (M/F)	9/11	10/10	11/9

$p > 0.05$.

Table 2. Basal cardiovascular parameters. Values are expressed as mean (SD).

	Group 1	Group 2	Group 3
Systolic arterial pressure; mmHg	128.7 (2.18)	128.3 (1.98)	128.8 (2.19)
Diastolic arterial pressure; mmHg	88.1 (2.29)	88.0 (2.27)	87.2 (3.33)
Pulse rate; beat.min ⁻¹	91.5 (9.37)	91.1 (7.24)	91.8 (5.65)

$p > 0.05$.

Table 3. Mean diastolic arterial blood pressure (mmHg) at various time intervals in the three groups. Values are expressed as mean (SD).

Group	B	B ₁	T	BI	I ₁	I ₂	I ₃	I ₄	I ₅	I ₇	I ₁₀
1	88.1 (2.29)	90.3 (2.08)	88.8 (1.27)	91.0 (1.03)	102.3* (1.21)	97.5* (1.82)	96.7* (2.08)	93.8* (1.61)	91.3* (1.71)	90.1 (2.06)	89.0 (2.34)
2	87.9 (2.37)	86.1**† (2.29)	85.2**† (2.30)	87.2**† (2.30)	81.1***† (2.29)	79.1***† (2.29)	81.7*** (1.98)	82.6** (1.35)	84.5** (1.32)	85.2** (1.54)	86.9 (1.99)
3	87.7 (2.16)	80.1*** (2.29)	79.4*** (1.87)	80.4*** (1.31)	76.1*** (2.29)	76.3*** (2.25)	78.2*** (2.26)	78.9*** (1.75)	82.9*** (1.75)	84.9** (1.71)	86.7 (1.78)

* $p < 0.01$ within the groups as compared with the basal value.

** $p < 0.01$ between groups 1 and 2 and 1 and 3.

† $p < 0.01$ between groups 2 and 3.

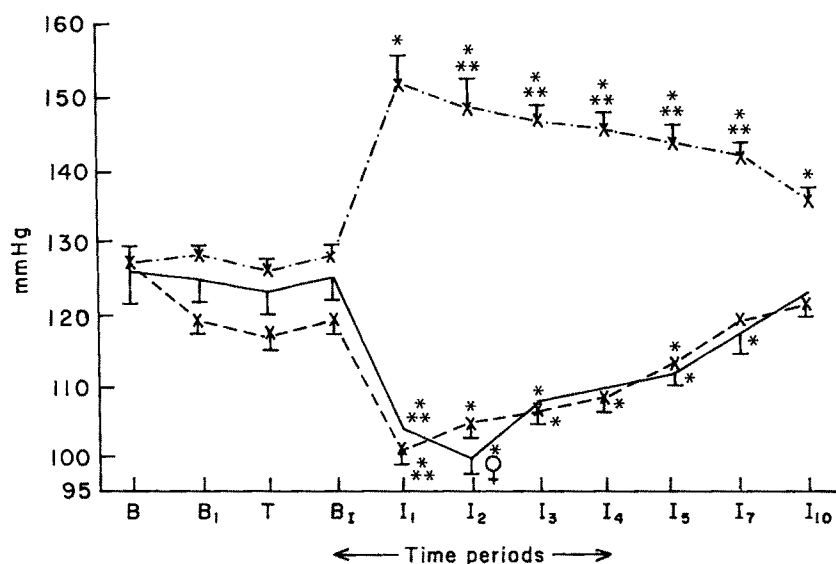


Fig. 2. Systolic arterial pressure in the three groups of patients at various time periods. —x—, group 1; —, group 2; —o—, group 3; * $p < 0.01$ within the groups vs basal; ** $p < 0.01$ between groups 1 and 2 and 1 and 3; $\circ$, $p < 0.01$ between groups 2 and 3.

bolus intravenous injection ($80 \mu\text{g.kg}^{-1}$) 45 s before laryngoscopy to attenuate the hypertensive response to direct laryngoscopy and tracheal intubation [24]. Nitroglycerin ointment has been used [25] for the same purpose, although drug absorption from the skin can be affected by many factors [26]. Intranasal nitroglycerin has been used, but the procedure described is cumbersome [13].

Isosorbide dinitrate buccal spray is a newer preparation and has been used successfully in the treatment of angina [20, 21]. The haemodynamic changes with the spray form occurred earlier, were more pronounced and lasted longer than those produced by the intravenous preparation. When a drug is used to attenuate the hypertensive response to tracheal intubation, the principal aim is to

match its peak effect to that of the stimulus. Isosorbide dinitrate is detected in the plasma within 15 s after the administration of the spray and reaches a peak within 5 min [20]. It acts by dilating the capacitance and resistance vessels, with the consequent reduction in the work load and oxygen consumption of the heart. This is the first study in which the spray has been used to obtund the cardiovascular responses to laryngoscopy and tracheal intubation.

The treated patients in groups 2 and 3 showed a significant decrease in systolic, diastolic and mean blood pressures following the spray, laryngoscopy and intubation when compared with the placebo group 1 patients, who had a significant increase in their systemic blood pressure. The duration of the decrease in systemic blood pressure

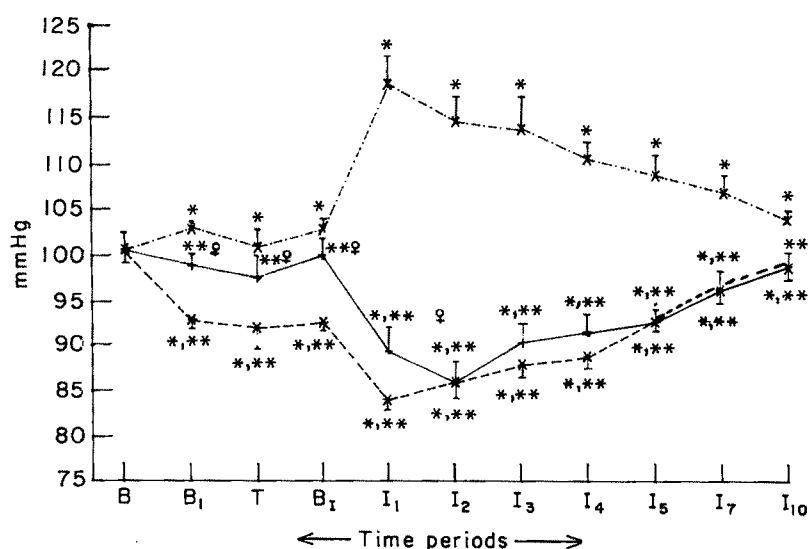


Fig. 3. Mean arterial pressure in the three groups of patients at various time periods. —x—, group 1; —, group 2; —o—, group 3; * $p < 0.01$ within the groups vs basal; ** $p < 0.01$ between groups 1 and 2 and 1 and 3; $\circ$, $p < 0.01$ between groups 2 and 3.

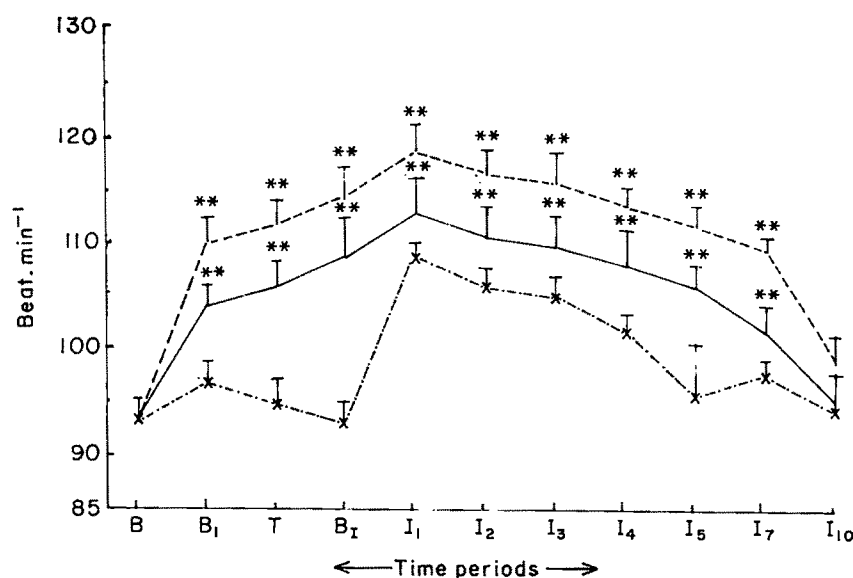


Fig. 4. Heart rate in the three groups of patients at various time periods. —x—, group 1; —, group 2; ---, group 3; * all values are significant ($p < 0.01$) within the groups vs basal in all groups, hence it is not shown in the graph; ** $p < 0.01$ between groups 1 and 2 and 1 and 3.

(10 min) was significantly greater in group 3 patients, in whom an interval of 150 s was allowed between the spray and the tracheal intubation, compared with group 2 patients (4 min) in whom the spray-intubation interval was 90 s. The use of the spray, however, did not abolish the tachycardia associated with laryngoscopy and intubation (Fig. 3).

Tachycardia is a feature of vasodilator agents and has been demonstrated previously with nitroglycerin and sodium nitroprusside [9, 13, 25]. However, the degree of tachycardia was of lesser magnitude with nitroglycerin ointment compared with isosorbide dinitrate spray or intranasal nitroglycerin [13]. None of the patients in the present study experienced the side effects of the drug. Normocardia was ensured in this study.

Apart from the suppression of the pressor responses to tracheal intubation, isosorbide dinitrate spray may benefit the ischaemic myocardium by improving oxygen delivery to the cardiac muscle [27–29].

In conclusion, we found that isosorbide dinitrate spray was a simple preparation to use for attenuation of hypertensive responses to laryngoscopy and tracheal intubation. It was effective when administered either 90 s or 150 s before tracheal intubation but the magnitude and duration of the decrease in systemic blood pressure was greater in group 3, in which 150 s had been allowed to elapse before intubation. However, since patients receiving isosorbide dinitrate spray had significantly greater tachycardia than those in the control group, consideration should be given to the additional use of a beta-adrenergic blocker in patients with conditions such as myocardial ischaemia and intracranial aneurysms.

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Predicting difficult intubation

Dr Frerk (*Anaesthesia* 1991; **46**: 1005–8) has encountered the major problem facing investigators in this field, which is that difficulty is actually quite rare. It has become conventional to regard only those cases classified as Cormack and Lehane grades three or four [1] as 'difficult' and there are only four such patients in Dr Frerk's study. Dr Frerk has included grade two patients in whom a bougie was used to

facilitate intubation in his 'difficult' group. I do not think that this should become part of the definition, since the threshold for employment of a bougie is very variable. Some people use a bougie on virtually every case. If we exclude the grade two cases from Dr Frerk's study, the incidence of difficult intubation becomes 1.6%, which is similar to previous findings [2–4]. Excluding the grade two

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cases means that the Positive Predictive Value (PPV, the proportion of cases predicted to be difficult, who actually were difficult) of the Mallampati test is only 7.6% and of the Patil test 7.5%. The PPV of the combination is 29%. These values of PPV for the Mallampati and Patil tests are similar to those found by Oates *et al.* [3] for the Mallampati (4.4%) and Wilson Risk Sum (8.9%) [2].

I believe that Dr Frerk is right to say that tests must be quick to perform and also reliable. Unfortunately, the evidence so far is that they are not particularly reliable, but they must be better than nothing and can be made easier to perform. In the case of the Mallampati test we should adopt Mallampati's own description of only three classes of pharyngeal visibility [5]. A fourth class was inserted by Sansoom and Young [6], perhaps in the hope that they would correspond to the four grades of glottic visibility described by Cormack and Lehane. Such precision is not justified in a test which is notorious for observer error [3, 7]. Patil used a wire gauge and Dr Frerk a pencil to measure thyromental distance, both of which instruments can be mislaid. Fingers are a satisfactory substitute, three of my fingers being about 6.5 cm in width.

I believe that difficult intubation can be more successfully predicted by using the principles suggested by Drs Brechner and Block [8, 9]. They pointed out that exposure of the glottis depended on forward protrusion of the mandible and adequate movements of the cervical spine, particularly extension of the head at the cranio-cervical junction (CCJ). Protrusion of the mandible depends on the proper function of the temporomandibular joint (TMJ) and this can be classified by asking the patient to push their chin as far forward as possible. There are three degrees of protrusion. If the lower incisors can be protruded anterior to the upper incisors, this is position A. If the upper and lower incisors can touch, this is position B, and if the lower incisors cannot be brought forward to touch the upper incisors, it is C. Flexion/extension movements at the CCJ can be assessed by a method suggested to me by Dr D. Menon. The patient is asked to maximally flex the cervical spine and the examiner's hand is placed on the back of the neck to immobilise further movements. The patient is then asked to nod his head. The range of movement is assessed as normal or reduced, based on the examiner's experience. These tests (TMJ B, C and reduced CCJ movement), which could be regarded as a refinement of the Wilson Risk Sum, have proved superior to the Mallampati and Patil tests in a study nearing completion here.

However, the prediction of difficult intubation will never be free from false positives, because of the arithmetic of sensitivity and specificity when the incidence is as low as 1–2%. For example, with an incidence of 1% and a test of 95% sensitivity and 99% specificity, 51% of the positive results will be false. We must accept false positives and evolve policies that are acceptable to anaesthetists and patients. In particular, fiberoptic competence should be required for accreditation.

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A reply

Thank you for giving me the opportunity to reply to Dr Calder's letter. As he rightly mentions, difficult intubation is rare and to try to address this problem I included a retrospective study of 23 cases of known difficult or failed intubation which confirmed the sensitivity of the tests suggested. A prospective study would have to look at about 1500 patients (to gather as many cases.)

I accept his point that only Cormack and Lehane grades 3 and 4 cases are usually classified as difficult; the reason that I included some grade two cases was that it had proved impossible to intubate them without a bougie. In all these cases I felt it was worth recording this on the anaesthetic chart to warn future anaesthetists that intubation had not been straightforward. (There were many grade 2 patients in the study who were not classified as difficult.)

I strongly disagree with his suggestion that we should be using Mallampati's original description of three classes for the very reason he alluded to, that observer variation is such a problem. I agree with the recent suggestion of Tham *et al.* [1] that the test should be simplified to just two grades, easy or difficult, with a clear cut off point (as suggested in the summary and discussion of my paper).

I would agree that it is possible to lose a 7 cm marker, although I have had the same one for 2 years. Fingers are only a satisfactory substitute if they measure exactly 7 cm and we all have different sized hands, but anything known to be that length would be a suitable alternative. This could be a pen with a piece of sticky tape round it or even a £5 note which is 7 cm from top to bottom.

Looking at the two tests he suggests, they are both interesting. His measure of head extension focuses particularly on the upper cervical spine where restriction of movement is important, but the test is subjective, relying on observer experience. Thyromental distance, however, gives an objective measure of head extension and has shown itself to be sensitive in other studies [2, 3]. His other test, mandibular protrusion, is particularly interesting since it addresses a problem not tested by other means and is also objective. I look forward to seeing the details when published, to see if it can match the sensitivity of the combination of thyromental distance and Mallampati test, with as few false alarms.

The positive predictive value of the combination of tests is 64% if the difficult grade two cases are included, but even if they are excluded, as Dr Calder suggests, giving a figure of 29%, this would mean that expert help would be mobilised unnecessarily only twice for each time that it was needed. I think that that would probably be acceptable for most departments providing a fiberoptic intubation service.

Bellhouse and Dore [4] first suggested combining tests to give a predictive risk. Biebuyck's editorial on difficult

intubation [5] confirms my suspicion that the way forward in prediction is by combining tests and specifically mentions Mallampati and thyromental distance, the combination of which has also been shown to be useful in an independent American study [2].

I would thoroughly endorse his final sentiment that all anaesthetists should become competent with fiberoptic instruments and then false alarms (and indeed difficult intubations) will not pose the great problems that they do now.

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The laryngeal mask—a modification in its use and design

The laryngeal mask airway (LMA) has now established a place in anaesthesia although discussion continues regarding minor modifications in its use and design. Insertion of the mask may occasionally be difficult, particularly at the point where the LMA meets 'the resistance off the posterior pharyngeal wall' [1]. At this point the tip of the deflated mask may fold back as simulated by the mask on the left in the photograph.

In our experience, insertion of the LMA is facilitated if

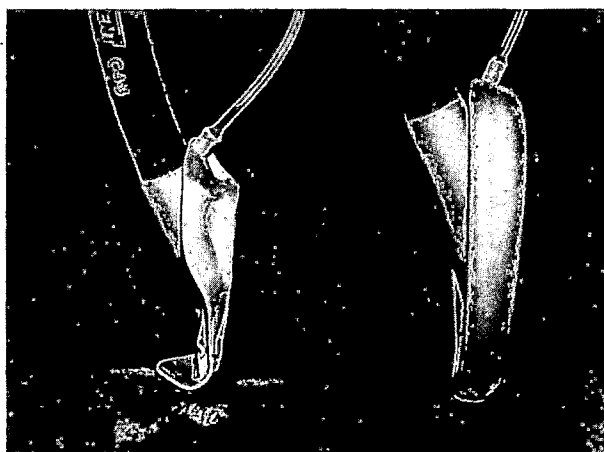


Fig. 1.

the mask is inflated to a low pressure, so that no dimples are apparent in its outline, but is readily moulded by the soft tissue of the oropharynx. This is depicted by the laryngeal mask on the right in the Figure; both masks are shown supporting their weight on their tip to simulate the effect of resistance acting on their tip during insertion. The partially inflated mask is readily inserted, need not be rotated, and once in position it is rarely necessary to inflate the mask further and then by only 1–5 ml. A partially inflated 'soft' mask may be readily removed postoperatively without trauma.

This offers the prospect of modifying the design of the laryngeal mask. If the range of the available sizes was expanded to include, for example, eight models, each with a mask of approximately 3 ml larger than the predecessor, then a mask of the correct size could be chosen for a patient so that the mask would require neither inflation for insertion nor deflation for removal. This range of masks would not require a valve or inflation line; they would be simpler, more durable and cheaper than the existing models.

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Use of the laryngeal mask airway in the presence of a bleeding diathesis

We would like to report a complication arising from the use of the laryngeal mask airway (LMA) in a patient with abnormal clotting function.

A 69-year-old man was admitted for treatment of a severe myelodysplastic syndrome and a Hickman catheter was inserted in the right subclavian vein to facilitate chemotherapy. He had a low platelet count, which was refractory to treatment with platelet concentrates due to the presence of antiplatelet antibodies, and continual bleeding from the operative site led, after 2 days, to a decision to re-explore the entry site. Anaesthesia was induced with etomidate, deepened with nitrous oxide, oxygen and isoflurane and a size 4 LMA inserted using the 'blind' technique described in the manufacturer's instruction manual, in the belief that this would cause less

trauma to the larynx than tracheal intubation. The airway was easy to introduce, and spontaneous breathing continued using the nitrous oxide/oxygen/isoflurane mixture. Monitoring consisted of ECG, automated noninvasive blood pressure (Dinamap), inspired oxygen concentration, pulse oximetry and end-tidal carbon dioxide. The patient's head was swathed in sterile towels and positioned on a head-ring, with the shoulders raised on a sandbag. During the course of the operation the head was moved several times by the surgeons to improve access to the operative site.

After 15 min, slight stridor could be heard, the movements of the rebreathing bag decreased and the end-tidal carbon dioxide started to rise. Oxygen saturation, however, remained high. After a few more minutes, as the

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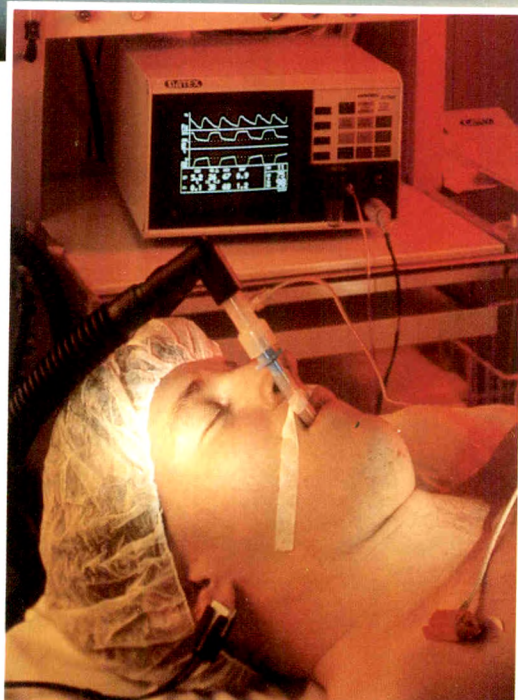
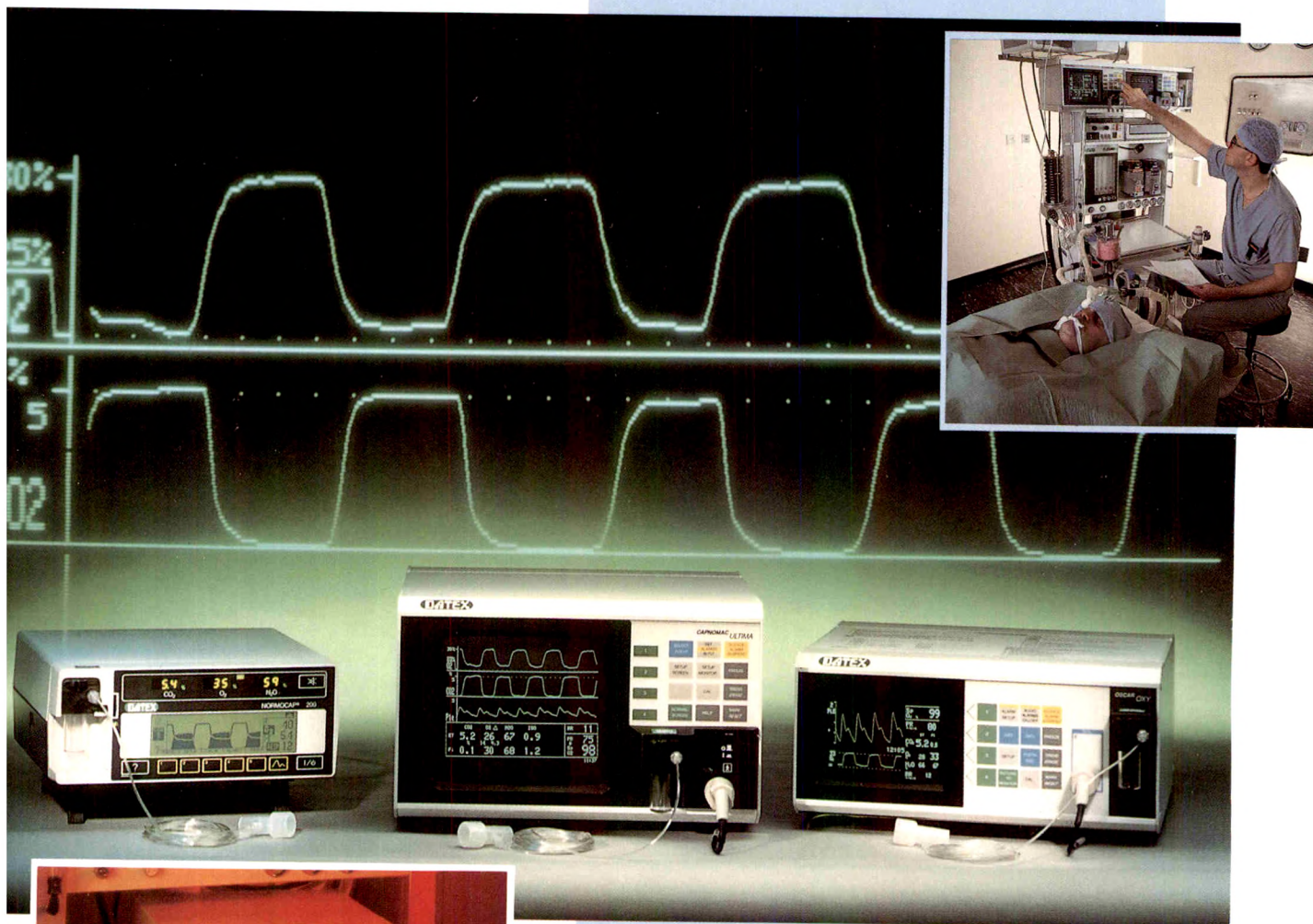
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stridor persisted, the surgeons were asked to stop, the drapes and LMA were removed. Laryngoscopy revealed a cherry-sized haematoma just above the right vocal cord and the patient's trachea was therefore intubated with a cuffed orotracheal tube and his lungs ventilated for the remainder of the operation. Repeat laryngoscopy at the end of the procedure showed considerable enlargement of the haematoma and this continued to enlarge over the next few hours. Extubation never became possible and mechanical ventilation was continued in the intensive care unit for some days. Because of his bleeding tendency and his laryngeal haematoma, chemotherapy for his underlying condition was never instituted; his condition deteriorated and he died some 4 days later without ever being able to maintain his own airway.

At the time, the LMA seemed the best way of maintaining the airway for this operation. Events subsequently proved this belief wrong and we now feel that the LMA should not be recommended for patients with uncorrected problems of haemostasis. Possibly for such a patient, provided there is no risk of airway contamination, a simple facemask technique would be the best approach, accepting the consequent slight impairment of surgical access. Alternatively, in patients at risk of airway soiling, tracheal intubation and ventilation using a high volume, low-pressure cuffed tube, might have been used with less risk of trauma than the LMA.

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Laryngeal mask misplacement—causes, consequences and solutions

The letter from Dr Southern *et al.* referring to suggested modifications to the LM ignores the need to form a very thin leading edge to the mask tip in order to avoid collision with the epiglottis during insertion. The folded-over tip is a natural consequence of failure to keep the mask pressed flat against the palate during insertion. Nandy *et al.* have shown [1] that the mask tip may also sometimes pass between the false cords to lie against the vocal cords. This is likely to happen if the mask tip is not kept pressed backwards during the entire insertion process. This can be achieved by pushing the LM all the way into place with the index finger maintaining a backward as well as downward pressure as shown in Figures 1 and 2. However, difficulty can be experienced in some cases and I believe Portex Ltd of Hythe, Kent are conducting a trial of a form of introducer tool which might be of value in preventing the tip from folding over, although it is by no means certain that this would prevent the misplacement noted by Nandy *et al.*

Thompsett and Cundy describe a haematoma in a myelodysplastic patient with a severe bleeding disorder, arising just above the right cord during operative exploration of a right subclavian wound which had continued to bleed for 2 days after insertion of a Hickman Line. The LM does not normally come into contact with the interior of the larynx and could not therefore cause the

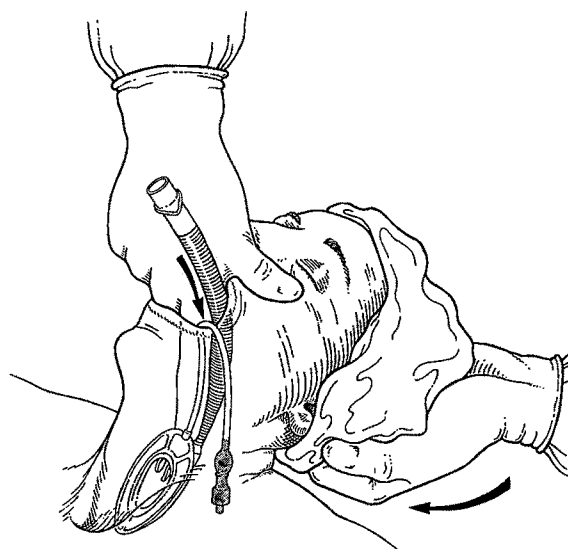


Fig. 2.

haematoma described, unless it was misplaced on insertion or became dislodged as a result of surgical manipulation, either of which may have occurred in this case. However, tracheal perforation by a needle tip may occasionally occur as a complication of insertion of a subclavian line, and the lesion described might therefore have arisen as the result of surgical manipulation opening up a needle track injury from the initial Hickman Line insertion, since this was on the same side.

The Instruction Manual does not refer to bleeding diatheses as a contra-indication to use of the LM, because it was considered that other forms of airway maintenance were likely to be equally or more liable to cause bleeding, since even a face mask may need to be accompanied by insertion of an oropharyngeal airway which may cause pharyngeal trauma. I do not believe the case described by Thompsett and Cundy should be used as an argument for making bleeding diatheses an absolute contra-indication to its use, but it does illustrate the need to be aware of possible LM misplacement and the importance of developing an insertion technique which makes its occurrence less likely. Misplacement of the LM tip between the false cords may be difficult to detect in patients breathing spontaneously with low flows, but becomes apparent if the lungs are inflated manually, when resistance to inflation, often with a sound resembling stridor or

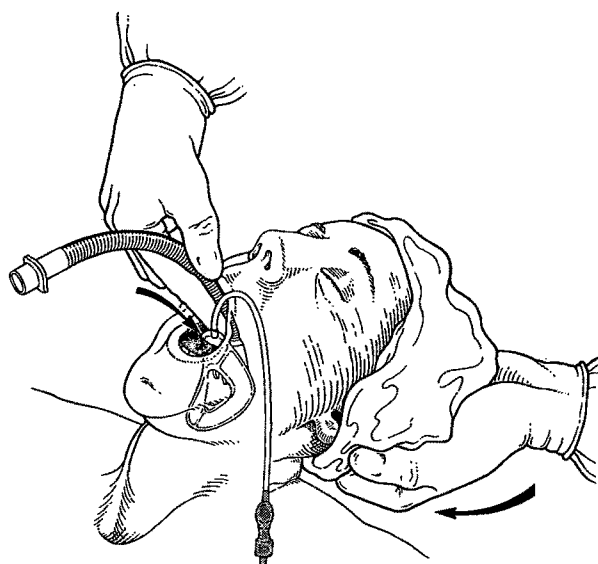


Fig. 1.

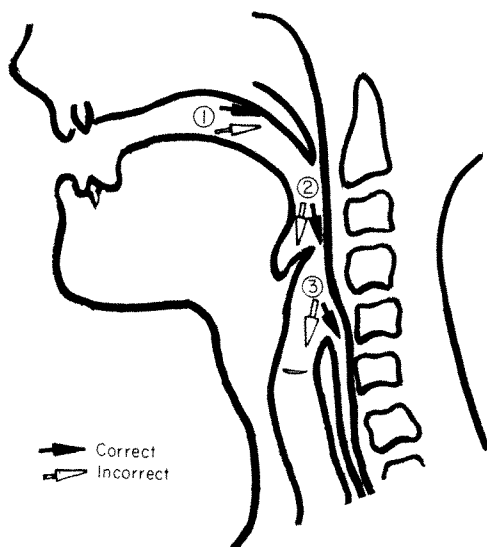


Fig. 3. The arrows correspond to positions of the mask tip during insertion.

bronchospasm, will occur. Another point to remember is that nitrous oxide rapidly increases the intra-cuff pressure during anaesthesia, so that these sounds may begin to occur spontaneously as the mask tip progressively enlarges within the laryngeal vestibule in cases where misplacement has occurred. Finally, the front of the neck should be observed after LM placement and during cuff inflation, to confirm that the cricoid prominence bulges forward, as well as the thyroid; if only the thyroid cartilage is seen to move forwards and if in addition the mask tube seems further out

of the mouth than usual, misplacement of the cuff tip between the false cords is likely. Sometimes it is possible to feel an abnormal increase in resistance to cuff inflation when about half the normal amount of air is injected; this again would suggest the cuff tip is misplaced.

The passage of the mask tip downwards into the hypopharynx during insertion is illustrated diagrammatically in Figure 3, in which the black arrows show the correct path and the white arrows the incorrect path which may be followed. It will be noted that there are three levels at which problems may occur. *Position 1*: the correct position of the mask tip, as it is guided downwards by the soft palate, is contrasted with the incorrect position where collision with the uvula or the posterior pharyngeal wall is about to occur. *Position 2*: the mask tip is closely applied to the posterior pharyngeal wall, so avoiding the posteriorly-lying epiglottic tip, while the inadequately deflated or incorrectly angled tip collides with the epiglottis. *Position 3*: The posteriorly-angled mask tip continues to point away from anterior structures, so avoiding entry into the larynx or collision with the arytenoids. Note that the final position of the mask tip in the hypopharynx should correspond with a level approximately between the 6th and 7th cervical vertebrae, some 3 cm deeper than that at which the misplaced tube tip would come to rest against the vocal cords.

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Use of the laryngeal mask airway during magnetic resonance imaging

The presence of ferromagnetic material in the proximity of the magnets during magnetic resonance imaging (MRI) may produce unwanted interference and distortion of the image produced. Thus it is important to avoid the use of metallic connectors in gas supplies and airway connectors when general anaesthesia and airway maintenance is required.

A 6-year-old, 21 kg child with a suspected Ewing's tumour of the right scapula, recently underwent MRI scanning and required general anaesthesia. Anaesthesia was induced with propofol and a size 2.5 laryngeal mask (LMA) was inserted. Maintenance of anaesthesia was achieved using a propofol infusion with spontaneous ventilation of 100% oxygen delivered by an Ayre's T-piece. The image that was obtained was distorted and we suspected the presence of unwanted metallic material. On inspection we discovered that the valve and pilot tube of the LMA were overlying the child's shoulder, and the valve appeared to contain metallic material. The valve was therefore removed, inflation of the cuff being maintained by knotting the tube, and the scan proceeded uneventfully with marked improvement in the image quality. The valve was X rayed later and the presence of a metallic spring was confirmed (Fig.).

The manufacturers, Colgate Medical Ltd, have informed us that this valve is now standard in *all* sizes of recently produced LMAs, including the size 2.5 which has only recently become available. This is in contrast to the older valve assembly that did *not* contain a metallic spring. It is important for anaesthetists to note this since the LMA may be used frequently for airway management during MRI

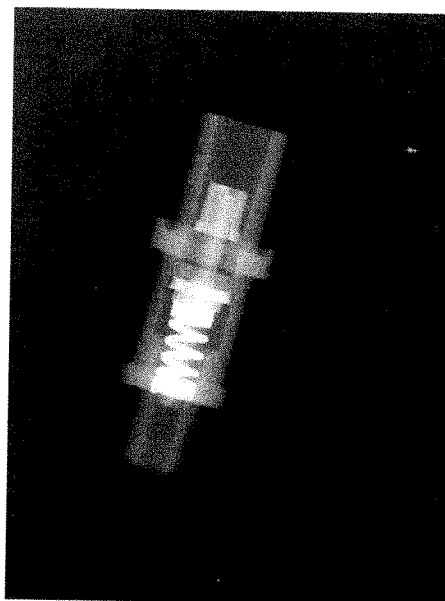


Fig. 1.

scanning. When the area under investigation lies within close proximity to the LMA, then distortion due to this valve assembly may be a problem.

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Identification of laryngeal mask airways

The fractured laryngeal mask airway (LMA) reported by Drs Crawford and Davidson (*Anaesthesia* 1992; **47**: 76) is similar to an incident reported by me (letter submitted). We also found it impossible to mark the laryngeal masks (LM) indelibly with any ink or paint. However, we have been able to engrave a simple code onto the hard plastic connector at the proximal end of the tube (Fig. 1). This is done using an industrial engraver (Powerline 172) and does not appear to damage the connector. The code is noted at each pass through the Central Sterile Supply Department and the LM replaced after 40 sterilisations. Since starting this routine we have had no further incidents with broken or failed LMs.

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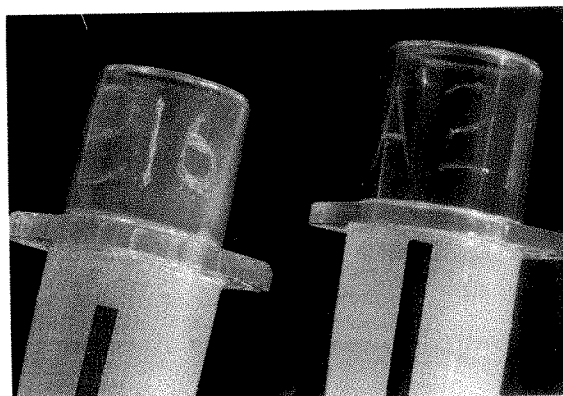


Fig. 1.

Is clonidine an anaesthetic in man?

We read with great interest the editorial focusing on α_2 -adrenergic agonists and clonidine by Professor Feldman and Dr Ooi (*Anaesthesia* 1991; **46**: 1003–4). There has recently been increasing interest in the peri-operative use of clonidine due to its sedative as well as other sympatholytic effects, and the emergence of clonidine in clinical anaesthesia has made critical evaluation of the properties of this class of drugs necessary [1]. The most prominent physiological action ascribed to α_2 -adrenoceptor activation is inhibition of neurotransmitter release [2]. This effect is accompanied by sedation, decreased arterial blood pressure and heart rate, and most importantly decreased sympathoadrenal activity. Thus, clonidine, as well as other α_2 -agonists, has a direct attenuating effect on the sympathetic nervous system. This in turn provides, for example, increased haemodynamic stability during surgery [3, 4].

In animal studies clonidine and such novel α_2 -agonists as dexmedetomidine have been shown to possess potent anaesthetic properties. In fact, dexmedetomidine, which is about 10-fold more selective towards α_2 -adrenoceptors than clonidine, may be a complete anaesthetic by itself in sufficiently high doses in certain animal models [5]. However, the anaesthetic properties of α_2 -agonists have not been investigated in man. Thus far, all human clonidine studies dealing with anaesthetic and/or narcotic requirements have involved almost exclusively haemodynamic end-points [3, 4]. α_2 -agonists reduce cardiovascular and sympathoadrenal reactions caused by noxious surgical stimuli, thus blunting the stress responses mediated by the sympathetic nervous system. Although the sympathoadrenal attenuation may be beneficial [6], the doses of α_2 -agonists required to reduce the autonomic responses may not be analgesic [7, 8] and may not provide anaesthesia *per se* in man. Thus, if α_2 -agonists are used in connection with combined balanced anaesthesia (with analgesics, intravenous or inhaled anaesthetics, and muscle relaxants), such autonomic nervous system reactions as increases in heart rate and blood pressure may not be useful as reliable signs of insufficient anaesthesia. Our

concern is that extensive use of clonidine or other α_2 -agonists as anaesthetic adjuvants carries a potential risk of awareness under anaesthesia.

In addition, this accentuates the complexity of the strategies required to investigate anaesthetic effects of a drug in man. This is especially pertinent if the drug has direct effects on the parameters used for the indirect assessment of the 'anaesthetic depth'. Thus, further carefully designed studies investigating the anaesthetic properties of α_2 -agonists in man are needed before widespread peri-operative use of this class of drugs can be advocated.

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Esmolol and clonidine—a possible interaction

An 80 kg man was scheduled for an elective gastrectomy. His hypertension had been treated with bendrofluazide 2.5 mg daily, and although the pre-operative blood pressure was 140/90 mmHg, there was ECG and chest X

ray evidence of left ventricular hypertrophy. Anaesthesia was induced with thiopentone and diamorphine and the lungs ventilated with oxygen, nitrous oxide and enflurane. Muscle relaxation was maintained with atracurium. Some

15 min after a bolus dose of clonidine 50 µg had successfully controlled persistent hypertension, he became tachycardic with rates of up to 170 beat.min⁻¹. Carotid sinus massage was ineffective and he was given esmolol 75 mg by slow intravenous injection. The heart rate decreased over the next 2 min to less than 20 min⁻¹. No pulse could be felt and cardiac massage was commenced; atropine 1.2 mg, adrenaline 1 mg, and 10 ml of calcium chloride were given intravenously. Within 5 min the blood pressure was 110/60 mmHg and heart rate stable at 110 min⁻¹. Surgery was recommenced and the patient made an uneventful recovery.

Esmolol has little intrinsic sympathomimetic activity and could conceivably have been entirely responsible for such profound cardiac depression, although in those published studies [1, 2] where the drug was administered to control intra-operative tachycardia, it was well tolerated. Clonidine attenuates sympathetic outflow and has been associated with bradycardia during anaesthesia, but we are not aware of any reported examples of this magnitude, and neither are the manufacturers. Since clonidine and beta-adrenoceptor blockers have formed a recognised combination for oral antihypertensive therapy, could an

exaggerated response result from intravenous administration? Kanitz *et al.* [3] observed significant hypotension when esmolol was given to a patient taking clonidine, and although this responded promptly to intravenous ephedrine 10 mg, the authors felt unable to recommend the combination until further evaluation had been performed, with which we would have to agree.

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Psychogenic seizures after general anaesthesia

A 29-year-old nurse, ASA 2, was admitted to hospital for termination of a 15-week pregnancy. She gave a history of intermittent fits since the age of 14 years, particularly associated with pregnancy. During one such episode she had lost consciousness suddenly whilst at work, and had remained unconscious for 6 days. Following this episode, she had been maintained on phenytoin, but she had not taken any anticonvulsants for 5 years following an assessment by neuropsychiatrists, who felt that her seizures were anxiety-based.

Termination of pregnancy was performed under general anaesthesia using thiopentone and halothane. Recovery was initially uneventful; however, 10 min after recovery she was witnessed having a series of violent generalized convulsions which were treated immediately with parenteral diazepam and chlormethiazole. Phenytoin and phenobarbitone therapy was also started. Despite this treatment, the seizures continued and it was only after infusions of thiopentone and alfentanil were commenced that good control of the fits was obtained. Subsequent respiratory depression required tracheal intubation and positive pressure ventilation. She remained free of seizures overnight, but fitting recurred on four occasions the next morning. She was therefore transferred to this hospital.

On arrival, she was fitting violently; between seizures she was flaccid and had an absent gag reflex. Intermittent boluses of thiopentone 200–300 mg, followed by a thiopentone infusion, were required to control the seizures. Phenytoin and midazolam were also given, and central venous pressure and direct arterial pressure monitoring were started. A cranial CT scan and a lumbar puncture were performed and showed no abnormality. A 24 lead electroencephalogram (EEG) was undertaken, but, despite a recording being made during a seizure, showed no evidence of epileptiform activity. In view of this and of her history, the thiopentone and midazolam were stopped, and

she woke up uneventfully and free of fits. A diagnosis of psychogenic seizures (pseudoseizures) was made, and she was discharged from hospital on a reducing dose of phenytoin.

Desai *et al.* [1] suggest that approximately 10% of patients presenting with intractable seizures have a psychogenic cause for their fits; such patients, as our case report shows, are often inappropriately treated with potentially toxic anticonvulsant drugs. The authors suggested four diagnostic criteria in such patients; deviation of seizure from characteristics of known seizure type, absence of epileptiform activity in the ictal EEG, absence of slowing in the postictal EEG, and lack of relation of seizure frequency to decreasing plasma concentrations of anticonvulsant drugs. Roy *et al.* [2] compared a group of patients with psychogenic seizures with a matched group of epileptic patients and found significant differences in respect of history of psychiatric disorder, attempted suicide, sexual maladjustment and a current affective syndrome. They suggested that patients with such histories presenting with seizures should be observed initially prior to institution of anticonvulsant therapy.

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T. PARRY
N. HIRSCH

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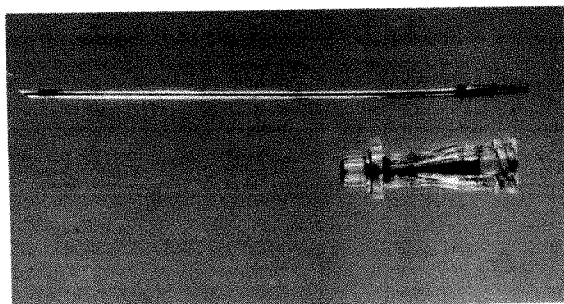
A possible complication with a sheath introducer

A 30-year-old woman was admitted to our intensive care unit following a laparotomy for perforated bowel. She was septicaemic and required the insertion of a pulmonary artery flow-directed catheter. The left subclavian route was

chosen and an Arrow Sheath Introducer Kit was used. The left subclavian vein was located without difficulty using the 18 gauge introducer needle. When the guide wire was threaded and the needle withdrawn, the hub separated

from the needle shaft, leaving the needle in the vein. Fortunately, the needle was just visible and was removed using a pair of forceps with no harm to the patient.

The introducer needle and separated hub were returned to the manufacturers who have informed us that the hub/needle connection is made by using an epoxy adhesive and there was residual adhesive found on the needle shaft indicating that the bonding process was not missed. Thus the bond should have been made between the needle shaft and the hub. Apparently, the bond formed was only marginal in strength. The manufacturers advised us that, for other reasons, they have now changed the material they use to manufacture the hub to a new one that gives better bond strength results.



This complication, although rare, could have very serious effects should the needle get lost in the vein. We recommend that before inserting any introducer needle into a central vein, the integrity of the hub/needle connection is inspected and tested.

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B. MATTA
S. WILLATTS

A reply

The most important criteria for the manufacture of any medical device is to make it safe and effective. This has been, and will continue to be, our constant goal. Although our records indicate this incident is extremely rare, with a frequency of less than 1 in 250 000, the action we have taken is expected to reduce further or eliminate this event. We continue to strive for problem-free products.

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P.J. MCGREGOR

The Oxford Miniature Vaporizer at high temperatures—evaluation of an external bypass

Field anaesthesia can be required at temperatures for which vaporizers were not designed; e.g. the 45–53°C measured in a Gulf War operating room [1–3]. Because data for the Oxford Miniature vaporizer (OMV) in the Triservice Apparatus have been published only from 13° to 27°C [4], isoflurane outputs of an OMV were measured at –1 to 55°C using a Datex Capnomac in a laboratory study at this hospital. The F_i values in Table 1 apply to gas sampled downstream from the Oxford ventilator used to generate drawover flow.

The further the temperature rose above about 30°C, the more dangerous the maximum OMV outputs became, but controllability was retained, as shown by near linear OMV calibration curves obtained at given temperature (–1 to 55°C), minute volume (2, 4 or 6 l) and pattern of flow (plenum or drawover). For plenum flows, and all calibration points, gas sampling was from just past the OMV. Calibration outputs were measured after 15 s at each control level setting. To reduce the need (as temperature rises) to limit vapour output by increasingly precise OMV setting, various external bypasses were tried, so that vapour laden gas would be suitably diluted by the bypass fraction, whilst maintaining the F_{iO_2} provided by fresh gas entering the now modified OMV.

The best bypass (Fig. 1) used two Bennett's connectors (CAT. 5959) joined at their narrow ends by Portex male and female tracheal tube fittings (100/252/100 and 100/251/001). The Bennett's wide ends were attached via corrugated tubing connectors (Airlife 001823) to the side arm of each

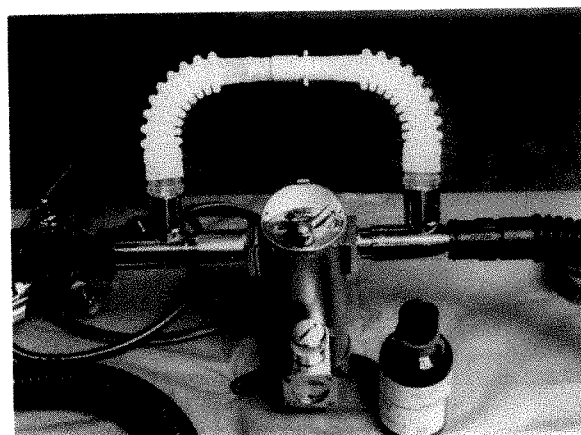


Fig. 1.

of two CIG bag mount adaptors (OBM 311388) fitted to the OMV inlet, and outlet.

OMV + bypass drawover calibration curves showed linearity and controllability of isoflurane output at 22 and 45°C for each minute volume, and together with the data in Table 2, confirmed the effectiveness of the bypass, even in the first few seconds of flow. Plenum calibration curves for this modification were nonlinear, and revealed excessive reductions of vapour output, especially at flows less than

Table 1. OMV drawover isoflurane delivery (F_i) vs temperature (T) and minute volume (MV) F_i values at 2 min, OMV set full on.

MV, l.min ⁻¹	T, °C	–1	8	22	34	45	55
2	F_i Isoflurane % v/v	2.3	3.6	4.7	9	12*	14*
6		1.9	3.2	5.0	6.6	10	10

Table 2. OMV full on + bypass at 55°C: drawover F_i vs time, and minute volume (MV).

MV, l.min ⁻¹	Time, min	$\frac{1}{2}$	1	2	6
2	F_i Isoflurane % v/v	5.2	5.6	5.5	5.4
6		3.6	4.8	4.6	4.2

Table 3. OMV + bypass: pressure decreases at 40 l.min⁻¹.

OMV	Bypass	Pressure (cmH ₂ O)
Full on	Absent	-3
Full on	Clamped	-3
Full on	Patent	-1.8
Occluded	Patent	-3

6 l.min⁻¹ Table 3 shows pressure decreases associated with drawover air flow of 40 l.min⁻¹ (1.2 l in 1.8 s) at 22°C through the OMV, with or without the bypass. Pressures were measured with a water manometer (clear plastic tubing of 5.5 mm internal diameter) attached to a T-piece at the ventilator intake. Similarity of the Table 3 values was predicted by the 55°C outputs in Tables 1 and 2, and by the drawover calibration data, but not by plenum data.

In conclusion, at temperatures from about 30 to 55°C, the specified external bypass reduced desirably the

otherwise excessive isoflurane drawover output of the OMV, but was unsatisfactory for plenum OMV use. Before any 'home-made' external bypass for the OMV is employed clinically, it should be tested by vapour analysis (and checked by the pressure drop technique), at the temperatures, minute volumes and plenum/drawover flow patterns in which it is to be used.

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Secondary transfer of patients

We are grateful for the reply of Vyvyan *et al.* (*Anaesthesia* 1992; **47**: 73-4) to our letter concerning the 'secondary transfer of patients'. We suggested that helicopters equipped to the minimum standards, as recommended by the report of the working party on such matters [1], possess insufficient monitoring to undertake secondary transfers. In reply, Vyvyan and his colleagues correctly comment that the standards document covered both primary and secondary transfers of patients; however, it did not distinguish between the equipment deemed necessary for each type of transfer. Indeed, it did not mention that a capnometer and facilities for invasive pressure monitoring should be included in the minimum standards required for secondary transportation of patients. The standards document will inevitably be used by the directors of helicopter systems as guidance for equipping such craft, and as such, should be more specific in its advice.

On the rare occasions that our hospital has been required to transfer patients by helicopter we have been offered a variety of craft, most of which were unacceptable in one way or another. In the incident referred to in our first letter, recognising the need to provide standards of care and monitoring equal to that in our own intensive therapy unit, we took considerable care to ensure that the helicopter in question would reach our own standards. Complete details of the patient's condition were discussed with the duty

flight registrar and he was made fully aware of our monitoring, ventilation and other equipment requirements. The transfer was accepted in the full knowledge of these. Furthermore, the medical director of the helicopter scheme in question is a member of Dr Bristow's working party and would therefore be fully aware of the standards recommended for the secondary transfer of an intensive care patient.

We agree with Dr Vyvyan and his colleagues that the craft used for secondary transfer must be appropriate for its intended role. However, it cannot reasonably be expected that a referring hospital, faced with an urgent need to transfer a critically ill patient and the offer of a helicopter service purporting to be adequately staffed and equipped, should be responsible for verification of the expertise of that system.

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Reference

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Timing of surgery in congenital diaphragmatic hernia

We read with interest the article regarding the timing of surgery in congenital diaphragmatic hernia (CDH) by Charlton *et al.* (*Anaesthesia* 1991; **46**: 820-3), reporting their experience with 86 neonates with CDH presenting within 6 h of life, and demonstrating significant stabilisation of 57 of the infants associated with delay in surgery and an overall mortality of 29%. While these results are certainly very encouraging, the authors should refrain from comparing their results to those of extracorporeal membrane oxygenator (ECMO) centres who use rigid criteria for the initiation of ECMO. Current ECMO criteria usually involve the use of the oxygen index (OI) (mean airway pressure $\times$ 100 $\div$ Pao₂) with an OI of greater

than 40 on three occasions as the most common criteria for the initiation of ECMO [1]. In the absence of ECMO availability, Bohn *et al.* have demonstrated, using the ventilatory index (ventilator rate $\times$ mean airway pressure) and the Pao₂, either before or, more reliably, 2 h after surgery [2, 3] that such indicators are very useful in selecting a population of infants with CDH having 90% or greater mortality. Others have defined infants with CDH as responders or nonresponders on the basis of the ability to achieve a postductal Pao₂ of greater than 13.3 kPa or a Pao₂ approaching normal [4, 5]. Subsequent reports have concluded that these indicators are inaccurate when ECMO is available, and that no infant should be denied support

using ECMO on the basis of any of these criteria [6-10], although one recent report describes blood gas and ventilation criteria which the authors consider to be predictive of death even with the use of ECMO [11].

If Charlton *et al.* wish to conclude that ECMO is of no benefit or would have only benefited a small number of their patients, then they should attempt to demonstrate that their infants with CDH were at least as ill and satisfied the criteria which have been demonstrated in the past to be associated with a high mortality. While the authors have referenced the use of the alveolar oxygen gradients and the use of the ventilatory index (their references 7 and 8), they have not provided any information in their paper that would allow the reader to compare the severity of the infants described using such indicators. The authors should be encouraged to provide this information if they wish to conclude that their results represent a significant improvement from other current experiences.

Charlton *et al.* also consider that a lung weight less than 35% of predicted excludes the possibility of ECMO, presumably because this is believed to indicate lethal pulmonary hypoplasia. We do not know how to diagnose lethal pulmonary hypoplasia, other than in an infant who dies despite the use of ECMO, and we would consider those six infants with <35% predicted lung weight as ECMO candidates.

We agree with the conclusion of Charlton *et al.* and many other groups that pre-operative stabilisation in congenital diaphragmatic hernia is beneficial, although a recent report from Liverpool found no difference in outcome between early and delayed surgery [12]. We wish to emphasize that these discussions and comparisons concerning the merits of the timing of surgery and use of ECMO will only be useful if authors provide blood gas and ventilation data so that the severity of the cases described can be compared to the published indicators.

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A reply

There appears to be no measure of severity of congenital diaphragmatic hernia (CDH) which has so far met with widespread acceptance and it remains to be seen whether any of the various predictive indices will permit a genuine comparison between centres with both markedly different approaches to treatment and differing populations. We arrange immediate transfer of all CDH babies in our region, undertake prolonged pre-operative medical treatment and aim to deliver all babies with an antenatal diagnosis in our own hospital. In contrast, some ECMO centres appear to take predominantly patients who are selected by survival of delivery, transfer to a paediatric surgery facility, early surgery and later removal to the ECMO centre. These contrasting systems present different problems in the application and timing of severity assessments. Lack of complete data from a referring unit may lead to a patient's 'best effort' being missed. Predictive indices have no place in the actual management of our patients (since we have decided not to offer ECMO). However, they are of interest and their relationship to the outcome of delayed surgery is under investigation.

There seems to us little doubt that some CDH patients have insufficient lung to survive on (as low as 15% expected weight in our series). ECMO is not a cure for pulmonary hypoplasia. We have no means of knowing exactly the minimal lung weight compatible with survival, but since Drs Finer and Etches appear to be advocating ECMO for every baby dying from CDH (as now do other groups [1-3]) since the predictive indices are considered unreliable) they should be able to tell us whether the lung weights of babies dying in the 'ECMO era' are now lower than those upon which our estimate of 35% was based [4]. We also await with interest a demonstration that the availability of ECMO has indeed altered the overall mortality from CDH in the communities served by the ECMO centres.

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A serious complication of minitracheotomy

We are grateful to Dr Clarke and his colleagues at St George's for reporting one of our more infamous ITU cases (*Anaesthesia* 1991; 46: 1041-3). However, we would welcome the opportunity to add some information which may explain how the apparently impossible did in fact happen.

The surgeon responsible was a locum consultant who, as far as I know, had never inserted a minitracheotomy before. In our hospital all minitracheotomies are usually put in by the anaesthetic staff, initially under supervision by one of two consultants, until skilled in the procedure. The patient was severely hypoxic on the ward and the anaesthetic registrar was called to assist in her management.

As sputum retention was thought to be the problem and being skilled in the procedure, the registrar inserted a minitracheotomy, removed the introducer and the device was used for suctioning. Before an X ray was possible, the patient's condition deteriorated further and she was transferred to ITU. A respiratory arrest occurred and the minitracheotomy was used to inflate the lungs with oxygen with concomitant improvement of oxygen saturation.

As tracheal intubation was deemed impossible, an urgent tracheostomy was required and the locum surgical consultant attempted to perform this on the Intensive Care Unit, removing the minitracheotomy at what was supposed to be the latest stage possible. On realizing that he could not insert the tracheostomy tube quickly, the surgeon asked for another minitracheotomy, which he inserted through the well of desaturated blood, towards the trachea. He suggested connecting oxygen to the device but the anaesthetic registrar found that ventilation through the device was impossible and it was discarded rapidly. Another attempt was made to insert the tracheostomy tube, this time successfully.

No one recalls seeing the introducer after this time and as no chest X ray was taken between the two minitracheotomies, it is not possible to say definitely on which occasion the introducer was left in. As the first was put in under relatively controlled conditions by an experienced registrar, it would have been instinctive to remove the introducer and its removal was confirmed by the nurse assisting. In addition, the minitracheotomy must

have been in the trachea on the first occasion as sputum was aspirated and saturation improved on ventilation. If the introducer had penetrated on this occasion the damage would have been seen in the trachea at subsequent fiberoptic bronchoscopy performed 2 days later. The other minitracheotomy was put in under 'panic' conditions by an inexperienced operator and was probably not in the trachea as evidenced by an inability to pass any oxygen through it. I feel this account lends support to the author's contention that the skill of the operator is crucial to the complication rate of this procedure.

The case also exhibits the fact that something so obvious and yet so bizarre can initially be missed on an X ray. An unkind twist of fate had decreed that the introducer had come to lie in such a position that on AP film it looked absolutely straight (see original article) and in no way resembled the familiar curve of the introducer.

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J. HAMMOND
B. BRAY

A reply

Thank you for letting me reply to Dr Hammond's letter. I wish to stress that our report in no way intended to criticise Dr Hammond or his ITU colleagues, and I apologise for any offence that may have resulted.

Our case does, however, illustrate the occasional difficulties a regional centre such as ours can have in deciphering a complete case from a referring hospital. Might I suggest that in future to avoid discrepancies between the hospitals, a copy of the case report be sent to the referring hospital and that their comments along with the draft copy be submitted to the editor. I would like to point out that in this case Dr Hammond was sent the final report for comment prior to submission.

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J. CLARKE

Expiratory obstruction from coincidence of sizing

The new operating theatres in Portsmouth recently opened equipped with an Anaesthetic Gas Scavenging System (AGSS) conforming to BS 6834: 1987. A transfer system (from circuit exhaust valve to the anaesthetic machine

mounted receiving system) is in use, manufactured by Ohmeda to BS 6834: 1987 standards. At the inlet end of the transfer system is a 30 mm female conical connector (Fig. 1) designed to mate with a 30 mm male circuit exhaust

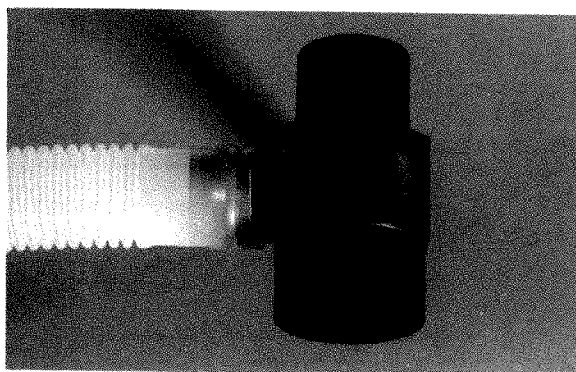


Fig. 1.

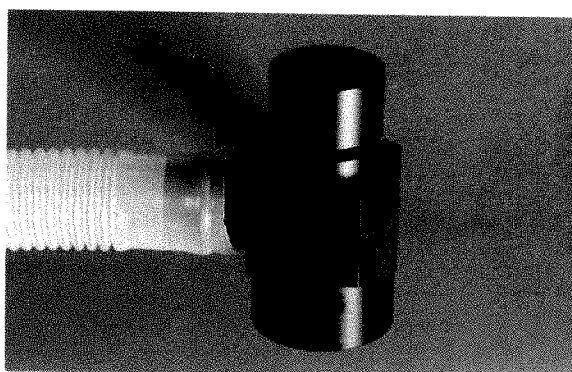


Fig. 2.

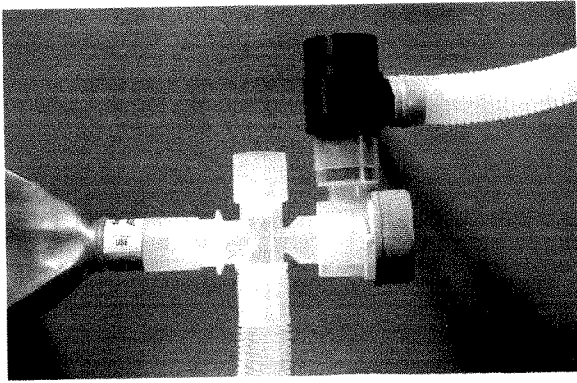


Fig. 3.

valve, mounted opposite to a positive pressure relief valve (Fig. 2).

Prior to commencing a routine operating list the anaesthetic machine in the induction room had been set up by the anaesthetic assistant with a disposable Bain-type circuit manufactured by Intersurgical. The transfer system had been attached to it. On testing the circuit it became rapidly obvious to the anaesthetist, that there was complete expiratory obstruction, even when the exhaust valve of the system was fully opened. Examination of the system revealed that the transfer system had been connected back to front, with the positive pressure relief valve firmly seated inside the 30 mm male exhaust valve connector (Fig. 3) producing complete expiratory obstruction. Reversal of the transfer system (Fig. 4) produced complete relief. This unfortunate coincidence of size of components produced by two different manufacturers, could have resulted in a dangerous expiratory obstruction.

This incident illustrates the importance, when introducing new equipment or circuitry, of proper education of all personnel as to the design and function of its components. Possibly, equipment manufacturers when designing components, could take into account the perverse ingenuity of the human race (among which are numbered anaesthetists and their assistants) to make mistakes if it is in any way possible to make them, and produce the nonfunctional surface of connectors in a nonround shape so as to prevent such accidental misconnections. One existing example of this is the outer surface of the female

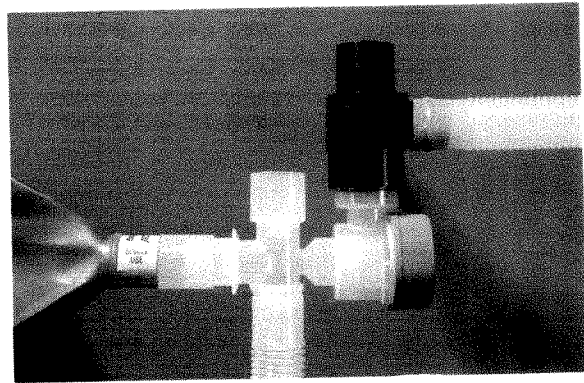


Fig. 4.

connector on the Newton Paediatric Valve for the Penlon Nuffield 200 ventilator. Likewise, components such as the pressure relief valve on the Ohmeda transfer system, which are in a position where they could be mistaken for a breathing system connector should possibly be deliberately machined square.

The necessity of testing breathing systems as well as anaesthetic machines is also reinforced by this event.

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J.R.A. MOON

A reply

Ohmeda thank the Editor for the opportunity to reply to this letter.

We endorse Dr Moon's comments with regard to the importance of educating all users when introducing new equipment and connecting equipment of different manufacturers, together with the checking of all equipment before use. In the case described, the instruction in the equipment Operation and Maintenance Manual together with labelling of the Ohmeda valve were provided to prevent the likelihood of misconnection.

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R.T. BRIDGES

Anaesthetic techniques and trainee anaesthetists

We would like to comment on the paper by Mulholland and Weir (*Anaesthesia* 1991; 46: 777-9) on a survey of anaesthetic techniques used by trainee anaesthetists in N. Ireland for elective surgery on children over 1 year of age. We wish to point out that we were not involved in, or informed of, the preparation of this paper and the views expressed therein are solely those of the authors.

In their paper the authors compare the techniques used by trainee anaesthetists who have had experience at the Royal Belfast Hospital for Sick Children with those who have no such experience. We were therefore surprised and disappointed to read that only one third of trainees who had previously worked at this Hospital visited their patients pre-operatively. This is contrary to our teaching and practice and we cannot understand why so many trainees apparently omit what is a vital part of anaesthetic care.

We welcome the finding that a high percentage of trainees use regional and local analgesia techniques for

postoperative pain relief. However, we would point out that we do not practice or teach the use of spinal or intercostal nerve blocks for this age group. Spinal anaesthesia is used solely for ex-premature babies requiring inguinal hernia repair. In this hospital children receive comprehensive electronic monitoring on a routine basis. However, this must be complemented by close clinical observation and a precordial stethoscope. We were therefore disappointed to read that the stethoscope was used so infrequently by trainees.

Like many other paediatric anaesthetists we believe that halothane remains the most suitable volatile anaesthetic agent for children. It is our custom to teach and practice repeat halothane anaesthesia without stipulating a time interval between exposures provided there has been no history of jaundice or unexplained pyrexia following a prior exposure. The authors advocate, amongst other things, the presence of parents at induction of anaesthesia. Although parental presence in the anaesthetic room is our

own routine, we believe that it is for the individual anaesthetist to decide how best to conduct his anaesthetic practice.

While surveys of this nature can be helpful and informative, we believe the interpretation of the findings may be of limited value unless wider consultation and advice is sought.

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P.M. CREAN
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R.H. TAYLOR

A copy of this letter was shown to the authors of the paper concerned, but no reply has been received.

Ed.

Pain relief following dental extractions

Garrioch *et al.* (*Anaesthesia* 1991; **46**: 828–32) state: 'The pain associated with multiple extractions and other surgical procedures in the mouth provides a useful model with which to study the analgesic properties of peripherally acting analgesics and anti-inflammatory drugs'. However, it is surely not ethically acceptable to continue studies like this where patients are used as animal models when adequate forms of pain relief, as outlined below, are readily available, especially when the authors conclude that nonsteroidal anti-inflammatory drugs given orally are not an efficacious means of providing analgesia following elective oral surgery.

In this unit we believe that adequate pain relief can be achieved using intra-oral injections of bupivacaine 0.5% prior to the commencement of surgery, with oral analgesics in the postoperative period. Several studies using a similar technique, including those by Chapman and Ganendran [1] and Tuffin *et al.* [2] have shown a significant reduction in postoperative pain with a corresponding reduction in analgesic requirements.

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References

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A reply

Thank you for the opportunity to reply to the above letter. We applaud the use of local anaesthetic intra-oral injections prior to the commencement of surgery for oral procedures and we agree that this may be an efficacious means of providing postoperative analgesia. It is not,

however, the only acceptable way of treating postoperative dental pain and its use is not currently standard practice amongst all oral surgeons in the UK.

It may not be necessary. This was indicated by one of the studies quoted in the communication (Tuffin *et al.*) which showed no statistical difference in pain scores 4–6 h postoperatively, irrespective of whether local anaesthetic infiltration had been used or not. The significant reduction in pain score at 24 h postoperatively is difficult to explain pharmacologically as any local anaesthetic effect would presumably no longer be applicable, although we accept the validity of the observation. Interestingly, Tuffin *et al.* did not administer any analgesic drugs in their control group until the postoperative period; thus it is not surprising that these patients were uncomfortable and that local infiltration was shown to be superior! In addition, local anaesthetics are known to be relatively inactive in the presence of infection, which is sadly often present in association with impacted wisdom teeth. For these reasons, the standard use of local anaesthesia may be justified, but is by no means an absolute requirement.

We believe our patients, far from being 'animal models' subjected to 'not ethically acceptable' treatment, received above-average standards of care. Fentanyl was used to attenuate the adverse surgical stimuli associated with tooth extraction. Frequent and sensitive assessment of postoperative comfort was made and analgesic drugs, which had previously been shown to be efficacious, albeit in other situations, were administered. Rescue analgesics were liberally used in the 30% of patients that needed them and thus satisfactory analgesia was guaranteed. We had no means of knowing that the drugs would be so poorly absorbed and, in fact, our work is unusual in displaying a plasma-drug concentration curve. This demonstrates the importance of measuring these plasma concentrations before drawing conclusions about oral analgesic drug effects and helps to explain why our regimen for oral administration was largely unsuccessful.

In conclusion, we accept that our results were disappointing. We wish to emphasise that we believed that our patients were receiving satisfactory treatment based on the numerous other studies to which we refer. The fact that our results did not support these previous data makes it all the more meaningful. In no way do we consider our work unethical.

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M.A. GARRIOCH
G.J. WARDALL

Craniotomy and PEEP

We wish to report a clinical problem arising during the use of the MIE Cavendish 680 M anaesthetic machine, and its attached MIE Carden ventilator.

A 57 year-old woman was undergoing craniotomy for removal of a large temporoparietal tumour. CT scanning

showed there to be a considerable amount of cerebral oedema together with midline shift. At craniotomy the surgeon complained that 'the brain was bulging'. This was in spite of slight head up position, no kinking of the neck veins, satisfactory gases (P_{aO_2} 16.9 kPa and P_{aCO_2} 3.4 kPa),

peak inflation pressures of 18 cmH₂O, and full muscle paralysis using a vecuronium infusion (zero post tetanic twitch count). Anaesthesia was being maintained with 67% nitrous oxide in oxygen, with 0.4% isoflurane, with boluses of fentanyl as required. She was haemodynamically stable, with a blood pressure of 100/60 mmHg and a pulse of 70 beat.min⁻¹. She received 150 ml of 20% mannitol intravenously, with a resultant 700 ml diuresis, but with no improvement in operating conditions.

The question was raised as to whether the obligatory 2.8 cmH₂O positive end expiratory pressure (PEEP) delivered by this ventilator was contributing to the poor operating conditions, as it may increase intracranial pressure [1]. The ventilator was changed to a Cape Waine Mark 3 with zero end expiratory pressure, with an almost immediate reduction in the brain bulging through the craniotomy.

It should be borne in mind that modern ventilators in which PEEP is inherent may be unsuitable for anaesthesia for craniotomy, as the reduction in venous return can result in unsatisfactory operating conditions, particularly when cerebral compliance is already poor.

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R.A. CHUNG
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Reference

- [1] AIDINIS SJ, LAFFERTY J, SHAPIRO HM. Intracranial response to PEEP. *Anesthesiology* 1976; **45**: 275.

A reply

Thank you for the opportunity to reply to the letter from Drs Chung and Fawcett. I agree with their last paragraph and endorse the fact that modern ventilators with in-built PEEP may not be suitable for anaesthesia for craniotomy.

We were surprised that our machine was in a situation where the in-built PEEP would be a problem. It is clear from both our user manual and the patient system pressure gauge on the machine that there is an obligatory PEEP of 2.8 cmH₂O. Also, during our discussions with the hospital staff, when the machine was first demonstrated by our local Area Sales Manager, the subject of PEEP was discussed as this was considered undesirable in certain cases. As the PEEP is an integral part of our design we were unable to reduce this to zero and advised the hospital accordingly.

Having taken steps fully to advise the hospital staff on the use of our equipment we regret that the reported incident occurred.

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T. LONGMAN

Conventions in imaging

The paper on septic atrial thrombus following central venous catheterisation by Dr Joshi and colleagues (*Anaesthesia* 1991; **46**: 1030-2) is interesting because it illustrates the important place of different imaging techniques in diagnosis. However, several points should be made: (1) from the configuration of the echocardiogram image, there is an assumption that it is an apical 4-chamber view although this is not stated by the authors. If this is the case, then the labelling of the atria and ventricles is incorrect; the ventricles should lie towards the top of the image. The conventions in displaying cross-section ultrasound images are that, just as in a chest radiograph, the left side of the image corresponds to the right side of the patient. The anatomical site and plane of the examination should be stated. (2) As reproduced, the echocardiogram image does not show a convincing

abnormality. (3) The white cell scan image is puzzling; there is no indication of the orientation of the view (e.g. anterior, posterior or lateral) and the normal landmarks used in interpretation, characteristic distribution of liver and splenic uptake, are not present. As shown, the image does not enable one to draw any definite conclusions.

Meaningful dialogue between specialties needs an understanding, not only of the vocabulary of the subject, but also an acceptance of the same standards of presentation as is current in specialist journals. Where images are displayed, review by a radiologist would achieve greater comprehension with mutual benefit.

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R.J. DAVIES

Propofol and abreaction

Over the years a variety of drugs have been used in combination with psychotherapy for deliberate abreaction techniques and occasionally the anaesthetist may be asked to advise or assist colleagues from the psychiatric department in the administration of intravenous agents for this purpose. Until now, barbiturates such as thiopentone and methohexitone have been favoured with varying degrees of success. Abreaction as an unwanted side effect on awakening from propofol anaesthesia has been recorded recently [1]; however it appears that the use of propofol for elective therapeutic abreaction has not been described. We wish to describe an unusual recent case where intravenous propofol together with psychotherapy was used with some success.

A 14 year-old-boy of Indian origin was referred to the

child and family mental health team for consultation. For the previous 6 months he had been mute and 'unable' to use his legs to an extent that he had become wheelchair bound. Previously healthy, he had first presented to hospital with an acute episode of headaches, vomiting and slurred speech, which later resolved. A viral cause was suspected but extensive investigation for infective or neurological causes produced no positive result. When loss of voice and apparent loss of use of his legs subsequently supervened, a psychological basis for his condition was proposed, but initial psychiatric consultations produced little progress. The possibility of his having suffered some traumatic event was considered and through family consultations, agreement was eventually reached between patient and parents that he should undergo a session of

pharmacological abreaction in the hope that an insight might be gained into the origin of his disorder.

The technique was planned on a 'one-off' outpatient basis and in view of the good recovery associated with propofol a decision was made to use this. Small incremental doses of propofol were slowly given to the patient to a total of approximately 125 mg, the dose titrated against his degree of sedation so that he was kept drowsy, but still just able to nod his head in response to the questions and prompting of two therapists skilled in adolescent and family psychiatry. After a period of time the patient began slowly to include answers by whisper and eventually came to describe recent significant past events, underlying hostilities, loyalties and violence both within his immediate family as well as with relatives within the extended ethnic family group. It is possible that his 'condition' had been used by him, either consciously or subconsciously as a diversion in order to manipulate his family into focusing their attentions and concerns onto him, thus in the process alleviating pre-existing family tensions. Since then, with skilled follow-up and much counselling, he has improved so that he is now talking and walking again.

The use of propofol in psychiatry needs further evaluation, but unfortunately suitable opportunities are limited. However, if used in conjunction with psychotherapy, there are a number of observations and precautions to be made.

(1) As yet, abreaction is not a licensed indication for the

use of propofol.

(2) Abreaction is a technique likely to be carried out under the subdued lighting and relaxed ambience of the psychiatry couch. However, the usual anaesthetic precautions on administration of a powerful intravenous agent should still apply, i.e. administration of the drug by an experienced anaesthetist to a starved patient, an indwelling cannula, resuscitation facilities to hand and minimally obtrusive monitoring such as a pulse oximeter.

(3) Psychiatric recommendations include the recognition that clear consent needs to be obtained from all participants. Much work may have to be done to establish that the patient her or himself has made the choice. It is not a form of therapy that should be entered into lightly before other avenues have been explored beforehand.

There is the great need to ensure that the therapist has enough control over the situation to deal with a possible suicide risk that might be uncovered, and also that there is adequate skilled follow-up to ensure that the experience is made use of in further consultations.

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Reference

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Airway maintenance for short ophthalmological procedures in children

The modified Guedel airway described by Sethi *et al.* (*Anaesthesia* 1991; **46**: 1084) is in essence a Phillips airway with a Charles cap. This device was used in this hospital from 1968 to 1990 for ophthalmological procedures in children. We would suggest that, in current practice, neither device is now suitable for this purpose except for the shortest anaesthetics (up to 10 min). It is difficult to ensure correct positioning of the device and thus a clear airway for the duration of the procedure; the anaesthetist must frequently support the jaw and dilution of anaesthetic vapours requires high fresh gas flows which may increase pollution.

These problems may be overcome by the use of the laryngeal mask airway (LMA). We initially gained experience in the use of the LMA in small children for radiotherapy [1] and have since found the technique well

suited to ophthalmological procedures. The correctly positioned LMA provides a secure airway, leaving the anaesthetist's hands free. Satisfactory conditions can be obtained with oxygen, nitrous oxide and a volatile agent without the need for promethazine or pethidine and the children recover quickly. We now use this technique for the majority of children having ophthalmological procedures.

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Reference

- [1] GREBENIK CR, FERGUSON C, WHITE A. The laryngeal mask airway in pediatric radiotherapy. *Anesthesiology* 1990; **72**: 474-7.

Pain relief after day case laparoscopy

We were interested to read the article by Edwards *et al.* concerning pain relief after day-case laparoscopy (*Anaesthesia* 1991; **46**: 1077-80) since we have recently audited the figures for 1991 of our own Day Case Unit. Like the authors, we have found diclofenac to be unsatisfactory for analgesia after laparoscopic sterilisation, and intramuscular opioid analgesia is often required. However, we were surprised to read that pain necessitated overnight admission in seven out of 40 patients undergoing this procedure (17.5%) and we reject the suggestion that laparoscopic sterilisation is unsuitable for day-case surgery. In our unit, 275 laparoscopies were performed in 1991, of which 123 were for sterilisation. Of these, there were four admissions, two of which were for surgical reasons, leaving just two admissions for pain (1.6%).

We believe this is a very acceptable figure. However, we

now think we have solved the problem of pain relief after laparoscopic sterilisation by performing a bilateral mesosalpinx block as described by Smith *et al* [1]. This has proved remarkably effective and no patient has required intramuscular analgesia to date. A zero admission rate for pain would therefore seem to be an attainable goal for 1992.

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Reference

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$\dot{V}_{CO_2}$ following tourniquet deflation

Hoka and colleagues (*Anaesthesia* 1992; 47: 65–8) described the changes in oxygen uptake and carbon dioxide output on releasing leg tourniquets during orthopaedic surgery. Their study involved 23 awake patients undergoing lower limb surgery under spinal anaesthesia, and the results demonstrated increases in both parameters that were proportional to tourniquet time. We performed a similar study, measuring the increase in CO_2 output after tourniquet release in patients under general anaesthesia with controlled ventilation.

Twenty-five fit patients undergoing orthopaedic surgery to the lower limb were studied. Each patient was fasted, premedicated, and anaesthetised with a standardised technique involving thiopentone, vecuronium, fentanyl, O_2 , and N_2O . Ventilation was adjusted to maintain arterial oxygen saturation and end-tidal carbon dioxide values within the normal range. Anaesthetic depth and infusion rates were adjusted to maintain cardiovascular measurements close to the patients' norm.

Carbon dioxide output was measured over 5 min periods by collecting expired gases from the Manley Pulmovent ventilator in a Douglas bag and then measuring its CO_2 concentration by passing a sample through a Datex Cardiocap monitor. The volume of the 5 min specimen of expired gas was measured with a Wright's respirometer, allowing the CO_2 output per 5 min to be calculated by the following formula:

$$(\text{volume of sample}) \times \frac{(\text{mixed } CO_2 \text{ concentration})}{100}$$

A control reading was made during tourniquet inflation, and four consecutive 5 min samples were taken after deflation.

The mean tourniquet time was 72 min, with a range of 20–116 min. The mean pre-release CO_2 was somewhat lower than Hoka *et al.* found, reflecting the lower basal metabolic rate of the anaesthetised subject. In addition, this basal CO_2 output was found to correlate with age in an inverse linear fashion, with an r value of 0.7. Where our results differ is in

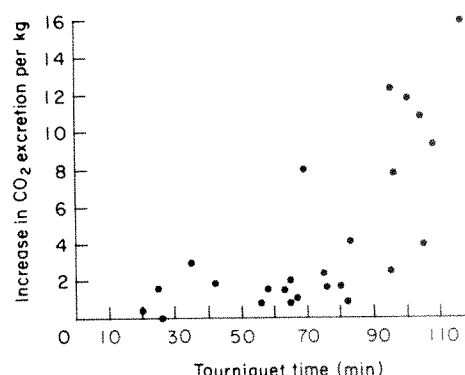


Fig. 1. Increase in CO_2 excretion per kg vs tourniquet time.

the relationship between tourniquet time and the 'excess CO_2 ' output on tourniquet release. Our results showed minimal extra CO_2 output with tourniquet times below 80 min. However, with longer tourniquet times, extra CO_2 output increased markedly (Fig. 1).

These results suggest that the metabolic impact of releasing a leg tourniquet after 80 min or less is unlikely to be of importance. However, with longer ischaemic times, the sudden entry of CO_2 and lactic acid into the circulation may affect the vulnerable patient, particularly if general anaesthesia prevents a compensatory increase in minute volume. Increasing the minute ventilation of paralysed patients for approximately 15 min after prolonged tourniquet times would, therefore, seem a sensible approach.

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M. DRESNER

I would like to thank Professor S. Feldman of the Magill Department of Anaesthetics, Westminster Hospital, for his help in performing this study.

Sufentanil and intracranial pressure

We would like to comment on the article by Weinstabl *et al.* (*Anaesthesia* 1991; 46: 837–40) on the effect of sufentanil on intracranial pressure (ICP) in neurosurgical patients.

The mild hyperventilation of all the patients in this study is not quantified in the article, so it is uncertain whether any cerebral vasoconstriction may have masked the effects of sufentanil on the cerebral vasculature. We believe that the title of this article may, strictly speaking, be incomplete. The authors appear to have omitted to consider any effect of residual midazolam. The elimination half-life of this benzodiazepine is 1–4 h and therefore stopping the midazolam infusion 1 h prior to the sufentanil boluses is an inadequate interval. Furthermore since midazolam is known to reduce cerebral blood flow, any residuum may have counteracted any contrary effects of sufentanil [1]. Therefore this study is actually examining the effect of sufentanil *plus* midazolam on intracranial pressure. We would also be interested to learn how the authors obtained informed consent from patients in a neurosurgical intensive care unit, some of whom had sustained head injury or intracerebral haemorrhage.

A recent abstract presented data suggesting that both sufentanil and fentanyl increase intracranial pressure in resuscitated head trauma patients [2]. Therefore the

controversy as to the effects of sufentanil on intracranial pressure remains and the article by Weinstabl *et al.* does not appear to resolve it.

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References

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A reply

We welcome the opportunity to reply to the comments of our article on the effect of sufentanil on intracranial pressure in neurosurgical patients.

Hyperventilation is quantified in the method section, stating 4 to 4.97 kPa.

We agree with the statement that the interval between stopping the midazolam infusion and giving sufentanil is shorter than the elimination half-life of the drug. Ethical considerations would not allow these patients to be without any sedation for longer than an 1 h. Furthermore we considered our interval long enough to study the effect of sufentanil on ICP. Cerebral blood flow (CBF) is not synonymous with ICP and CBF and ICP are not directly connected. ICP is dependent on the blood volume and not on the speed that it moves through the cerebral vessels. There may be some relationship between the clinical state and the drugs used.

Informed consent was obtained from the closest relatives of the patients.

In our view, no final conclusion can be made from the work of Bailey and coworkers [1]. Beside the use of

fentanyl and sufentanil, a variety of additional therapy (e.g. steroids, mannitol, midazolam and morphine) was given. Thus, the effects of sufentanil and fentanyl on ICP can be masked by a variety of drug-drug interactions.

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Reference

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Inappropriate filling of cylinders

Since the advent of our specialty, anaesthetists have endeavoured to evolve safety systems to ensure that cylinders and vaporizers contain the agent appropriate to the marking or colour of the container. In order to maintain the highest standards of clinical practice, systems such as the pin-index system, colour coded vaporizers and agent specific filling systems have developed often as a result of a tragic accident.

We have recently become aware of a method to circumvent such all important safety procedures, with regard to cylinders, which we feel we should communicate to other members of our profession.

The system to which we refer is a method of on-site refilling of size D cylinders from bull-nosed cylinders (size G and H) (Fig. 1). Size D cylinders are small and therefore ideal for patient transfer. However, being small they are rapidly exhausted; hence this system has been devised (see illustration). This method of refilling exhausted cylinders is wholly unsafe, as there are no non-interchangeable or pin-index system safeguards. This would appear to be in direct contravention of sections 4.1, 4.2 and 7.12 of the Health Equipment Information 163 (2/87) Code of Practice.

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P.M. WARD
M.W. PLATT

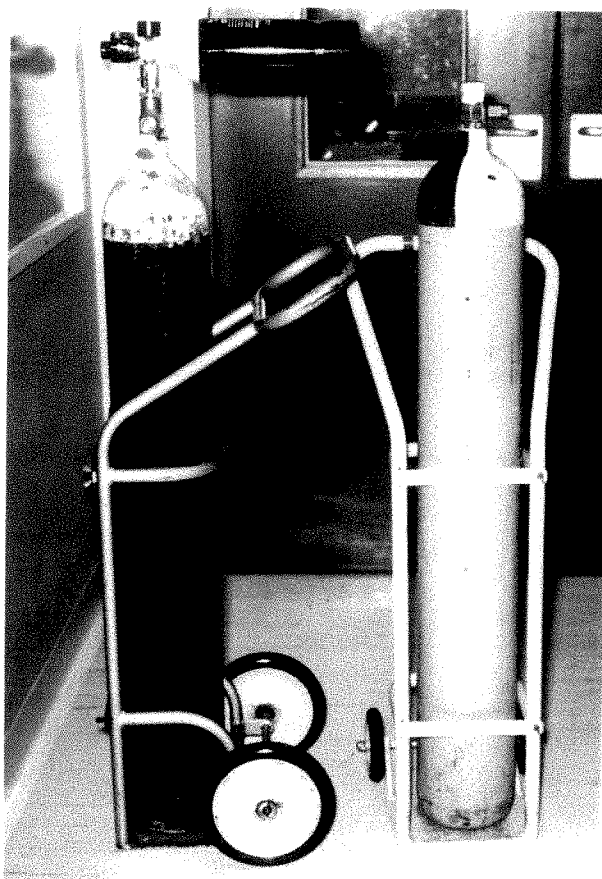


Fig. 1.

A case of malignant hyperthermia?

We were interested to read the case report 'A case of malignant hyperthermia?' (*Anaesthesia* 1991; 46: 942-5). However, we feel that there are some points which need clarification. Could the respiratory acidosis and tachycardia be a reflection of the anaesthetic technique in which the patient's lungs were ventilated for a period of 50 min using only an initial dose of suxamethonium (100 mg), 1% isoflurane and 0.15 mg fentanyl? The timing

of dantrolene administration appears to have coincided with the end of the surgical procedure.

We agree that the sustained high levels of CK and serum myoglobin are more indicative of muscle disease than malignant hyperthermia, in which the biochemical values return to normal very quickly.

This type of case exemplifies the need for diagnostic muscle biopsy and *in vitro* contracture testing (IVCT) in

etermining susceptibility to malignant hyperthermia. The omission of the detailed results of these tests leaves any conclusions regarding this case as conjecture. It should also be remembered that patients with other myopathies can have abnormal responses to the *in vitro* contracture tests [1] and therefore correct interpretation also requires details of any histological abnormality. Without this information we cannot comment on the true significance of the clinical events, although contrary to the authors impression, we feel this may *not* have been an MH crisis.

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Reference

- [1] LEHMANN-HORN F, IAIZZO PA. Are myotonias and periodic paralyses associated with susceptibility to malignant hyperthermia? *British Journal of Anaesthesia* 1990; **65**: 692-7.

A reply

The comments of Dr A.C. Christian *et al.* are interesting. However, we do not think that the clinical manifestations were caused by insufficient analgesia as they seemingly suggest. For this minor soft tissue operation, we consider the anaesthetic regimen used, i.e. N₂O/O₂ and isoflurane 1% and supplementary fentanyl, to be adequate. The precipitous increase in end-tidal CO₂, in combination with the rise in body core temperature over next 15 min and the progressive and persistent tachycardia are signs highly suggestive of malignant hyperthermia [1, 2]. We agree that the dantrolene administration coincided with the end of surgery. If the signs had been caused by inadequate analgesia they would have returned to normal immediately

after surgery. However, it took 30 min before the parameters returned to normal values.

We omitted the full details of the results of the diagnostic muscle biopsy and the *in vitro* contracture test (IVCT) because we felt they did not alter the conclusion: neurological investigations did not reveal signs of myopathy. The IVCT was equivocal, indicating that the patient should be regarded as susceptible [3, 4]. In the literature there is a general consensus that although the IVCT test is the most reliable at present, it is not ideal. Difficulties remain in the interpretation of the test, despite standardisation in its performance. Hopefully in the future more reliable noninvasive tests will become available.

We agree that histopathological investigations are important to diagnose myopathies, that are either associated with malignant hyperthermia [4] or that produce abnormal *in vitro* contracture tests. While our patient's biopsy was not normal, it was not diagnostic of myopathy. In conclusion, we still believe this patient suffered a malignant hyperthermia crisis. For your interest we are willing to forward details of the diagnostic tests to Dr Christian at his request and look forward to hearing his further comments in due course.

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References

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Red rubber Robertshaw tubes

We have recently noticed a change in the red rubber Robertshaw double-lumen tubes supplied through our Regional supplies network. Instead of the familiar Leyland Robertshaw tubes, we are now supplied with tubes made by Rusch Ltd., which superficially resemble Carlens and White's tubes, without the carinal hooks, although they are labelled 'Robertshaw'. On further enquiry, we discovered that Leyland Medical International Ltd ceased trading on the 24 May 1991.

The Rusch Robertshaw-type double-lumen tubes are *not* identical to those developed by the late Dr Frank L. Robertshaw in Manchester, in conjunction with the Leyland and Birmingham Rubber Company [1, 2]. We have noticed that they differ in several respects: (1) The Rusch tubes are based on the Carlens tube rubber extrusions, with their narrower lumina (Fig. 1). Their gas flow characteristics would thus resemble those of Carlens tubes, and the tubes would present more difficulties with tracheal and bronchial suction and the passage of a fiberoptic bronchoscope. (2) There are several differences in the right upper lobe orifice. That of the Rusch tube is shorter (15 mm compared with 17 mm in the medium sized tubes), narrower (3.5 mm compared with 5 mm), and widens less on inflation of the bronchial cuff. The Rusch tube has no inflating bronchial cuff proximal to the right upper lobe orifice; the Leyland tube has a short length of cuff to seal the right main bronchus (Fig. 2). These features (a design which originated with the Gordon-Green tube in 1955 [3]) have recently been shown to give the Leyland

right-sided Robertshaw tube the best margin of safety in positioning with respect to the right upper lobe bronchus [4]. (3) The pilot tube colour-coding of the Rusch tube is opposite to that of the Leyland tube. Robertshaw coloured the bronchial cuff pilot tube (although there was an error in some of the early tubes), whereas Rusch have coloured the tracheal cuff pilot tube, as in the Carlens tube. This has caused some confusion to users of Robertshaw tubes, and led to some difficulties during thoracic anaesthesia. (4) The Rusch tubes are latex-coated. This

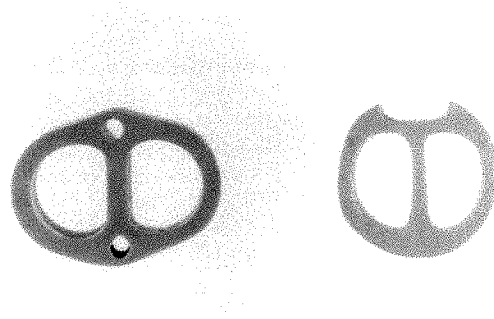


Fig. 1. Cross section of a medium Leyland Robertshaw tube (left) and a medium (37 Ch) Rusch Robertshaw (right).

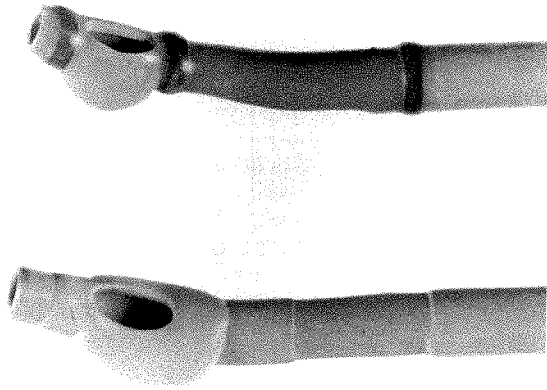


Fig. 2. Right bronchial cuff of a medium Rusch Robertshaw tube (top) and medium Leyland Robertshaw (bottom).

coating can become detached at the edges with use, and softens and becomes irreversibly sticky on accidental contact with organic solvents, rendering the tube useless. Leyland tubes are of uncoated, cured rubber.

We feel that the original Robertshaw pattern red rubber double-lumen tubes still have a place in thoracic anaesthesia, particularly for right bronchial intubation, and remain a standard against which others are compared. In centres using double-lumen tubes frequently, rubber tubes can still represent good value for money, if they are carefully washed and re-sterilised by staff taking precautions to prevent cross-infection [5]. Being re-usable and less toxic to dispose of than plastic tubes, rubber tubes could also appeal to the environmentally conscious anaesthetist.

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References

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A reply

The development of the Rusch Robertshaw endobronchial tube started in the late 1970s. When prototypes were available in 1980, these were sent to Dr Robertshaw for his evaluation and comments. He was impressed with the design of the tube, in particular the location of the cuff inflation channels, being positioned along the inner curve of the tube and not within the tube. He felt this to be an improvement on the original design. He had two further comments: that the 41 Ch model should have its total length increased by 5 cm; that the 'upper lobe' opening should be moved distally, so that the proximal portion of the bronchial cuff is proportionally more than the distal portion.

I would now like to address the points raised in Drs Lee and Phillips letter.

Gas flow characteristics. This is a function of tube size. The original Leyland design has an oval cross section; the medium size has a cross section of 16 mm/12 mm and

incorporates the cuff inflation lumens; the Rusch design has a round cross section with a diameter of 13 mm (size 37 Ch), which excludes the inflation lumens. The Rusch tube is available in three sizes 35, 37 and 41 Ch (OD), and if gas flow is the main selection criteria then a Rusch 41 Ch tube should be used.

We have not received any other comments regarding any potential difficulty in passing fiberoptic bronchoscopes or suction catheters through the Rusch tube. However, use of a smaller plastic suction catheter may resolve any suctioning problem. The authors are correct in their statement that the Rusch lumen construction is based on the Carlens tube.

Right upper lobe orifice. The differences in the right upper lobe orifice are correct. However, the tube was modified in 1989 to include a longer bronchial cuff that seals proximally to the opening. The opening was also moved distally so that the cuff could be made larger. From the figure supplied the tubes used by the authors were manufactured before these modifications were implemented (i.e. before April 1989). The Rusch tube has been in clinical use for 10 years and we have received, to my knowledge, no comments about a greater or lesser margin of safety being provided by a longer bronchial portion.

Pilot balloon colour coding. There is no international or national standards for colour coding of pilot balloons. The Rusch tube pilot balloons are the same colour, but the inflation tails are colour coded to match the respective cuffs; red for bronchial, cream for tracheal. With the use of re-usable tubes it should be standard procedure to test inflate the cuffs prior to use, to ensure that they are patent after sterilisation/cleaning. At this time any difference in pilot balloon colour and therefore which cuff is inflated should be noted.

Latex coating. A latex coating is a standard feature of all Rusch re-usable tracheal and endobronchial tubes. This gives the tube a smooth surface and makes it easier to clean. It also has the added advantage of causing less trauma to the tracheal mucosa than the old rubber cuffs. However, as with all rubber and latex products they should not be allowed to come in contact with organic solvents. If this is suspected the product should be destroyed immediately.

The Rusch Robertshaw tube has a number of design features that are superior to the original design. Radio-opaque ring markings on the proximal and distal ends of both cuffs enable confirmation of tube position by X ray. The latex cuffs are a one piece design and are free of seams inherent in the manufacturing method of the rubber cuff.

Rusch values the input of all clinicians and has an active Research and Development department and is very willing to consider any design modifications that improve product performance. After consideration of the points raised by the authors, we will be modifying the Rusch Robertshaw design to include: smoothing the relatively sharp edges of the bronchial opening; flattening and lengthening the tip of the bronchial branch; printing of the pilot balloons to identify the respective cuffs.

As the Leyland Medical tube is no longer available, we welcome the opportunity to discuss with any potential users of the Rusch Robertshaw, the slight difference in design and performance to help overcome any potential problems.

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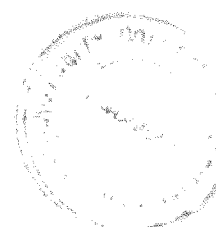
Safety Action Bulletin

Heated humidifiers when used with lung ventilators: risk of excessive temperatures SAB(92)12

Excess temperatures have resulted from humidifiers as a result of the control settings of the associated ventilator. Some ventilator designs include a standby mode and an overpressure safety device that cuts off or reduces the gas flow to the breathing system. If these are used in systems containing a heated humidifier, the flow through the humidifier may be reduced below that specified by the humidifier manufacturer. This may result in gas in the humidifier reaching excessive temperatures which will be delivered to the patient once normal operation of the ventilator is resumed/started.

Heated humidifiers: correct positioning and security of temperature monitoring probes SAB(92)13

Most humidifiers use a temperature probe situated at the patient outlet and although they may feel secure, may not be fully pushed home. This results in erroneous temperature readings being displayed on the humidifier with delivery of gas at an excessive temperature. Most heated humidifiers have a warm up time specified by the manufacturer and during this time, audible and visual alarms are disabled. If measuring probes are incorrectly positioned, the delivery temperature may be in excess of the set temperature when the warm up period has elapsed without an adequate warning to the user.



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The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology. Recommended method for the determination of gamma-glutamyltransferase in blood. *Scandinavian Journal of Clinical Laboratory Investigation* 1976; **36**: 119–25.

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OSLER AG. Complement: mechanisms and functions. New York: Prentice-Hall, 1976.

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American Medical Association Department of Drugs. *AMA drug evaluations*, 3rd edn. New York: Publishing Sciences Group, 1977.

Editor, compiler, chairman as author

RHODES AJ, VAN ROOVEN CE, comps. *Textbook of virology: for students and practitioners of medicine and other health sciences*, 5th edn. Baltimore: The Williams & Wilkins Co., 1968.

Chapter in book

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Agency publication

National Center for Health Statistics. Acute conditions: incidence and associated disability, United States, July 1968–June 1969. Rockville, MD: National Center for Health Statistics, 1972. (*Vital and health statistics, Series 10: Data from the National Health Survey*, No. 69) [DHEW publication No. (HSM) 72–1036].

OTHER ARTICLES

Newspaper article

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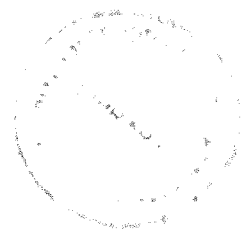
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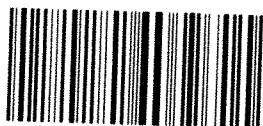
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Volume 47 Number 7 July 1992

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Editorial

Resource management

'When you can measure what you are speaking about, and express it in numbers, you know something about it. But when you cannot measure it, when you cannot express it in numbers, your knowledge is of a meagre and unsatisfactory kind.'

Lord Kelvin

We have suffered for years in the Health Service from our reactionary culture—from a reluctance to examine tools that have been shown in other areas to improve the delivery of a service, and to adapt them to our needs.

A past Minister of State for Health, Kenneth Clarke, introduced the most radical reform of health care delivery with his White Paper, accompanying it with an exhortation to prove that we needed more resources, not just clamour so: we could not. We did not know what to measure, or how to measure, and we did not have the tools with which to take measurements of either work or resources. Looking back, we had only the haziest idea of the financial framework in which we operated, and finance was far removed from treating patients.

Many will feel that we are now at the other extreme, when finance dictates practice to a quite unacceptable degree; it may well be that the present enthusiasm for knowing 'the price of everything and the value of nothing' will settle down once the methodology has been worked out, and we can all play the game comfortably again with revised rules. But without question a number of valuable lessons will have been learned about the way we should manage our resources.

It has taken politicians to point out to us that the scandal of patients being put onto waiting lists to wait years has to stop; it has been felt necessary to introduce a patients' charter, which states nothing that we would not expect for ourselves as patients. There is a clear need to define quality of practice, and check it through audit. Happily, management has taken to this as enthusiastically as medical staff. There is a cost attached to quality, but if we clearly define our goals and the resources necessary to achieve them, then the debate can be held sensibly.

There is now in the NHS a continuous cascade of 'purchasers' and 'providers'—each provider being a purchaser from the next tier. Thus departments of surgery are providers of services to health authorities and GP fundholders, and purchasers of services from anaesthetic departments. An anaesthetic department is a provider of services to departments of surgery, and a purchaser of drugs, equipment and other services.

Service departments should negotiate the quantity and quality of service directly with those to whom its service is provided. They are often near-monopoly providers, and are both strengthened and weakened thereby: on the one hand, being able to lay down the standards of service they expect to provide, which purchasers 'have' to accept, but on the other providing the quality of service that purchasers are willing to fund.

The key to much of the goal of improved quality lies in two concepts that are powerful tools, the 'care profile' and the 'business plan'.

The care profile is a description of how we intend to care for our patients, and can be as varied as each individual makes it—the primary purpose being to explain to those paying what they will get for their money. For anaesthetists, this may start with a pre-assessment clinic, describe the resources needed for a pre-operative round, and the investment in peri- and postoperative care that improves patient recovery. Postoperative pain control has often been a ritual intramuscular prescription, but the benefits of pain control teams and patient-controlled analgesia have been measured in financial terms, and the cost of improved quality willingly assimilated in many units. An extra quarter of an hour setting up an epidural in theatre costs about the same as the patient sitting 3 h in the ward, and will therefore pay for itself many times if the patient recovers faster; the care profile for the hip replacement may then include the extra time involved, and it is most unlikely that purchasers will object to the explicit cost of the improved quality.

The business plan, tying in tightly with care profiles, is designed to replace the endless competition for inadequate resources with structured development. As an example, if all patients should have oximetry and noninvasive blood pressure measured in recovery, and there are no instruments to do it, then the plan might describe their introduction, funded as an integral part of the operating theatre charges, and justified by a reduction in postoperative complications; such monitoring will cost barely one pound per patient, and is likely to pay for itself in savings in recovery staffing costs. The introduction of tighter controls on junior doctors hours and waiting list initiatives requires careful thought about staffing levels and proper costing in a business plan if a proper service is to be provided at all times. Purchasers and providers have to be able to inform themselves of the likely financial consequences of their schemes, and the key to successful negotiations by them is to have accurate information.

The question of costing and pricing treatments must be more intelligently assessed; there is a fundamental difference between firstly, the cost of providing a facility (overhead cost), whether it be an instrument or function, secondly, that of using it (marginal cost), and finally the price that includes money for investment and developments. The boundary between these is not sharp and there is no ideal way to combine them when pricing.

An operating department has an overhead cost of providing facilities, whether they are used or not, and then the marginal costs of servicing lists. The importance of the distinction lies in the fact that halving a particular activity will not halve its cost; in an operating suite, probably about half the income may be considered as overhead, providing a background to the

service, and the other half attributed to surgical activity. Thus if a planned activity is halved, the costs will be reduced by only a quarter. Conversely activity can be increased by a fraction considerably more than the fractional increase in cost.

We have been slow to appreciate the overwhelming importance of length of stay in hospital as a determinant of costs of treating patients. When a day in a general surgical ward might cost of the order of £200 almost any equipment, investigation or treatment that prevents morbidity and shortens length of stay is likely to be cost effective; our specialty has made its contribution by championing day surgery. A drive to look at ways of caring for patients other than in hospital by boosting community services to a far higher level than at present is long overdue: we cannot be party to a repetition of the scandal associated with the discharge of the mentally sick into an unprepared community, but one can buy a great deal of community nursing and physiotherapy for the cost of a hospital bed.

So, as we start to understand more about the mechanisms and costs controlling health care delivery, we discover the need for information systems to support our new culture. Fortunately, technology has just about arrived at a stage where it can provide the information services to support some of the principles involved.

The first principle is that clinicians 'own' their data; only then can they control its accuracy, have some degree of privacy, and review what is passed on up the line to clinical directors and managers. This implies a certain degree of computer power locally, holding databases that are currently in use, tailored to individual needs. Processing them locally to provide audit information will be routine; privacy is important in order that

staff can admit to practice shortcomings without fear, although subject to the Data Protection Acts.

The second principle is that the data collection process must be seen to be both practical and relevant; if it is not, then the information derived is unlikely to be of value. Fortunately, modern technology provides solutions to the practical problems of tedious data collection in the form of bar code and optical mark readers, for example. If the data produces useful information in the form of log books and clinical audit reports there may be enthusiasm for the process; if the data vanishes into the maw of a management mainframe, disenchantment may set in.

As we become clearer about the measurements we should make and what they mean, we will want to make comparisons. To do this, we have to agree the premises on which the datasets are collected, and how the data should be processed: only then are we comparing like with like—whether this be departmental workload comparisons or the extraordinary range of operation costs (actually, prices) being published.

Resource management is about finding the right balance between quality, quantity and cost; it has to be closely integrated with audit to monitor that balance. Those of us responsible for introducing it have to be sensitive to the difficulties some will have in coping with the new corporate computer culture, and must be careful to distinguish sound advances in planning strategy from political gimmicks.

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Editorial notices

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Induction of anaesthesia with propofol using a target-controlled infusion system

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Summary

Sixty patients premedicated with temazepam were allocated randomly to receive an infusion of propofol designed to achieve and maintain a target blood concentration of 3, 4 or 5 $\mu\text{g.ml}^{-1}$. Induction time was measured from the start of infusion to loss of verbal contact. The success rate of inducing anaesthesia within 3 min of achieving the target concentration was 40% when the predicted target concentration was 3 $\mu\text{g.ml}^{-1}$, 75% when the predicted target was 4 $\mu\text{g.ml}^{-1}$ and 90% when the target was 5 $\mu\text{g.ml}^{-1}$. There were no significant differences between the three groups for time to loss of verbal contact in patients who were induced successfully within 3 min. There were significant reductions in arterial pressure 3 min after achieving the target concentrations within the groups but not between them. The frequency of apnoea and pain on injection was small in all groups. Selecting a target concentration of 5 $\mu\text{g.ml}^{-1}$ would successfully induce anaesthesia in the majority of patients premedicated with temazepam without major haemodynamic or respiratory side effects.

Key words

Anaesthesia techniques; infusion, computer.
Anaesthetics intravenous; propofol.
Pharmacokinetics.

Induction of anaesthesia by administration of a bolus dose of propofol has been reported to cause a reduction in systolic arterial blood pressure of 20–30% [1–6], possibly in a dose-dependent manner [7]. In addition, apnoea has been reported following this method of induction [8–10]. A more satisfactory technique may be to induce anaesthesia by infusion thereby decreasing the induction dose of propofol and reducing the incidence of side effects [11, 12]. In these studies, propofol was infused at continuous rates until induction was achieved. The aim of the present study was to investigate induction of anaesthesia using a system designed to achieve and maintain three target blood concentrations of propofol [13].

Methods

Sixty patients undergoing minor surgery were admitted to the study. All patients were of class ASA 1 or 2, between the ages of 18 and 70 years and weighing between 45 and 100 kg. Approval from the Hospital Ethics Committee and informed consent from the patients were obtained.

Patients were premedicated with temazepam 20–30 mg

approximately 1 h before induction of anaesthesia. Baseline measurements of heart rate, systolic and diastolic arterial blood pressure, and oxygen saturation were recorded in the anaesthetic room and each patient was pre-oxygenated for 1 min before induction. The patients were allocated randomly to receive an infusion of propofol which was designed to achieve and maintain a target blood concentration of 3, 4 or 5 $\mu\text{g.ml}^{-1}$. The initial, rapid rate of 600 ml.h^{-1} was infused until the predicted target concentration selected for that patient had been achieved. Thereafter, the infusion rate was calculated by the pharmacokinetic model [13] to compensate for removal of the drug by elimination and redistribution.

Induction time was measured as the interval from the start of the infusion to loss of verbal contact and failure of induction was deemed to have occurred if anaesthesia was not induced within 3 min of achieving the target blood propofol concentration. In these cases, the target was increased to 6 $\mu\text{g.ml}^{-1}$. Arterial pressure was measured at 3 min after achieving the target blood concentration and the frequency of apnoea, defined as a cessation of spontaneous respiration for longer than 10 s, was also recorded.

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Table 1. Demographic data of patients. Values expressed as mean (range).

Initial target concentration; $\mu\text{g.ml}^{-1}$	3	4	5
Male/female	14/6	10/10	12/8
Age; years	48 (21–74)	43 (18–61)	42 (20–69)
Weight; kg	68 (43–99)	71 (51–100)	65 (52–95)

Table 2. Percentage of patients anaesthetised successfully at each ($n = 20$) of the initial target blood propofol concentrations shown and the mean (range) anaesthetic induction times.

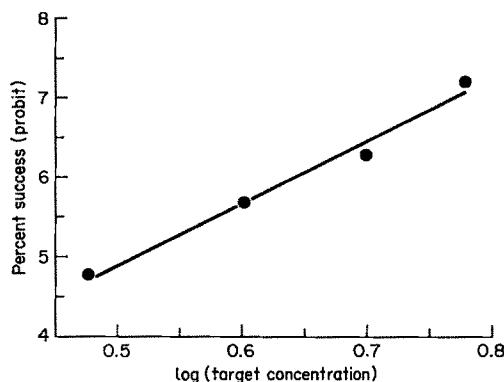
$\mu\text{g.ml}^{-1}$	3	4	5
Successful	40	75	90
Time to loss of verbal response; s	111 (90–170) ($n = 8$)	116 (75–184) ($n = 15$)	103 (45–136) ($n = 18$)

The results were analysed for statistical significance using analysis of variance and unpaired *t*-test for induction time, changes in heart rate and arterial blood pressure.

Results

The patients' demographic data are shown in Table 1. Anaesthesia was induced successfully without requiring a change of target in eight patients out of 20 (40%) when the predicted target concentration was $3 \mu\text{g.ml}^{-1}$, 15 patients (75%) when the predicted target was $4 \mu\text{g.ml}^{-1}$ and 18 patients (90%) when the target was $5 \mu\text{g.ml}^{-1}$ (Table 2). Probit analysis of these data demonstrated the dose-response of the target-controlled infusion system (Fig. 1). There were no significant differences between the groups in time to loss of verbal contact for patients who were induced successfully within 3 min (Table 2).

There were significant reductions in systolic and diastolic arterial pressure at 3 min compared with baseline values within each group but not between the three groups (Table 3). No significant changes occurred in heart rate in any of the three groups (Table 3). Two patients became apnoeic and three experienced pain on injection; neither side effect was related to the initial target concentration chosen.

**Fig. 1.** Probit analysis of the success rate in inducing anaesthesia and log (target blood propofol concentration) for 3, 4, 5 and $6 \mu\text{g.kg}^{-1}$.**Table 3.** Mean (SD) arterial blood pressure (mmHg) and heart rate (beat.min^{-1}) before induction and 3 min after for each ($n = 20$) target blood propofol concentration shown.

$\mu\text{g.ml}^{-1}$	3	4	5
Systolic before	134 (16.4)	130 (17.4)	130 (18.6)
At 3 min	116 (18.7)*	117 (14.1)*	114 (15.3)*
Diastolic before	80 (10.1)	81 (12.1)	76 (7.5)
At 3 min	72 (10.9)*	73 (14.4)*	66 (10.2)*
Heart rate before	75 (11.9)	81 (15.7)	79 (11.5)
At 3 min	75 (12.3)	79 (11.3)	78 (8.9)

* $p < 0.02$.

Discussion

Although the actual blood propofol concentrations achieved by the target-controlled infusion system would have been somewhat different from the values selected, they have been shown to be in close agreement for populations studied previously [13, 14]. The present study is the first to demonstrate the dose-response relationship of a target-controlled infusion system to induce anaesthesia.

There were significant reductions for all three groups in systolic and diastolic arterial pressures at 3 min compared with baseline values, but there were no significant differences between the groups. The mean decreases in systolic arterial pressure ranged from 10–13% and apnoea was rare. These figures represent an improvement compared with induction by bolus doses [1–6] and with continuous infusion [11, 12].

The process of induction of anaesthesia involves achieving the required concentration of drug at the effector site in the brain to produce loss of consciousness. A certain time is needed to transfer drug from the blood to obtain this effector site concentration and the time will vary with the concentration gradient between the blood and the effector site [15]. A high blood-effector site concentration gradient will reduce the time needed to induce anaesthesia, but may result in a greater frequency of adverse effects compared with a small gradient.

When anaesthesia is induced using bolus doses of propofol injected quickly, the high initial blood concentration will rapidly produce unconsciousness but may result in marked reduction of systolic arterial pressure and development of apnoea in a significant number of patients [1–6]. Reduction of the peak blood concentration when a slower speed of bolus injection [16] or slow continuous infusion [11, 12] is used leads to a longer induction time, but can reduce the frequency of adverse effects.

A target-controlled infusion is designed to achieve a predicted target blood concentration based on population pharmacokinetics and although the actual concentration may be greater or less than the target selected, it provides the closest approximation for any individual patient. The effect of the selected target concentration can be observed and, if insufficient, can be increased until the required endpoint is obtained [17]. Induction can therefore be achieved with less adverse effects, such as reduction of arterial pressure and apnoea, compared with administration of bolus doses and with a smooth transition to maintenance of anaesthesia [18].

Induction was considered acceptable by all patients even when induction was deemed to have failed at 3 min. The frequency of pain on injection was less than that reported

during bolus injection [16] and similar to that following induction by infusion [11]. The results suggest that selecting a target concentration of $5 \mu\text{g}\cdot\text{ml}^{-1}$ would successfully induce anaesthesia in the majority of patients premedicated with temazepam without major haemodynamic or respiratory side effects.

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Clinical evaluation of doxacurium chloride

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Summary

Doxacurium was administered to 50 adult patients for determination of potency ($n = 10$), onset and duration of clinical relaxation ($n = 40$). Cumulative dose-response showed the ED_{95} to be $33.24 \mu\text{g.kg}^{-1}$ (95% confidence limits 27.4–39.3). Doxacurium $33 \mu\text{g.kg}^{-1}$ was then administered to four groups of 10 patients each who had anaesthesia maintained with either fentanyl–droperidol or halothane and nerve stimulation carried out with single-twitch stimulation at 0.1 Hz or train-of-four stimulation at 2 Hz every 12 s. The onset and duration showed wide individual variation. The mean (SD) times to occurrence of maximal block were 8.5 (4.6), 6.1 (1.9), 6.7 (1.8) and 4.7 (1.3) min in the single twitch–fentanyl, train-of-four–fentanyl, single twitch–halothane and train-of-four–halothane groups respectively, although it ranged from 3.4 to 13.1 min in individual patients. The mean (SD) durations of clinical relaxation (recovery of single twitch or first response in train-of-four to 25%) were 65 (22.8), 52 (21.7), 70 (33.4) and 72 (21.0) min respectively with individual values ranging from 31 to 103 min. Although halothane administration increased the duration of clinical relaxation and train-of-four stimulation accelerated the onset of effect, the changes due to these were not significant. There were no adverse effects on heart rate or indirectly measured arterial pressure.

Key words

Neuromuscular relaxants; doxacurium.

Doxacurium is a relatively new non-depolarising muscle relaxant with a benzylisoquinolinium structure. It resembles pancuronium and tubocurarine in its duration of action, but is believed to be free from significant cardiovascular and histamine-releasing properties [1–4]. The present study was undertaken to estimate the potency of doxacurium in terms of its ED_{95} and to assess the onset, duration of clinical relaxation and reversibility during balanced anaesthesia and anaesthesia supplemented with halothane. The influence of the mode of stimulation on the onset and duration was also studied since this has been shown to influence the onset of action of other relaxants [5, 6].

Patients and methods

Fifty patients aged 18–65 years, of ASA grades 1 or 2, undergoing elective surgery requiring tracheal intubation and non-depolarising relaxant were included after obtaining written informed consent and approval from the University Ethics Committee. Patients with any neurological abnormality, who were more or less than 20% of their ideal body weight, had a family history of malignant

hyperthermia, who were pregnant, or taking any medication known to interfere with neuromuscular function were not studied.

Premedication consisted of oral diazepam 10 mg administered 60 to 90 min before operation. Electrocardiogram, noninvasive arterial blood pressure, end-tidal carbon dioxide concentration, peripheral oxygen saturation and skin temperature overlying the adductor pollicis were monitored throughout the study period in all patients. Ventilation was adjusted to maintain the end-tidal concentration of carbon dioxide between 4.5 and 5.5% and body temperature was maintained above 32°C. The study was carried out in two parts.

Part 1. Potency estimation

The first 10 patients were included in this part of the study. They were anaesthetised with thiopentone $4\text{--}5 \text{ mg.kg}^{-1}$, fentanyl $2\text{--}3 \mu\text{g.kg}^{-1}$ and 67% nitrous oxide in oxygen with additional doses of thiopentone and/or fentanyl as required. Neuromuscular block was monitored by percutaneous stimulation of the ulnar nerve with supramaximal stimuli of 0.2 ms duration at 0.1 Hz and the force of contraction of the adductor pollicis muscle recorded using

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Table 1. Age, weight, height and sex distribution. Values are expressed as mean (SD).

	ST/Fentanyl (n = 10)	TOF/Fentanyl (n = 10)	ST/Halothane (n = 10)	TOF/Halothane (n = 10)
Age; years	39 (10.1)	50 (12.5)	44 (14.9)	49 (12.9)
Weight; kg	66 (12.7)	69 (12.6)	66 (8.0)	68 (12.1)
Height; cm	167 (10)	167 (10)	164 (9)	167 (8)
Sex; M/F	4/6	5/5	4/6	5/5

ST = single twitch; TOF = train-of-four.

a force transducer and neuromuscular function analyser. Following stabilisation of control twitch height, doxacurium was injected rapidly in increments of $5 \mu\text{g.kg}^{-1}$, successive increments being given when there was no further reduction in twitch height in response to three successive stimuli, as described by Donlon *et al.* [7] and used previously in evaluating long-acting muscle relaxants [8, 9]. This was repeated until a reduction in twitch height of at least 95% was obtained.

The twitch height data were subjected to arc-sine transformation as described by Armitage [10] for responses involving the 0 and 100% points on the dose-response line. A log dose-response curve was constructed after regression analysis of the data and from which the ED_{50} and ED_{95} values were derived.

Part 2. Onset, duration of action and reversal characteristics

Forty patients included in this part of the study were randomly allocated to four groups of 10 each, according to whether they received increments of fentanyl, droperidol or 0.5–0.75% halothane for maintenance of anaesthesia and nerve stimulation using a single twitch (ST) at 0.1 Hz or a train-of-four stimulation (TOF) at 2 Hz every 12 s (Table 1).

The estimated ED_{95} as derived from Part 1 of the study was administered intravenously as a rapid bolus. The time to the start of the effect (lag time), onset of maximum block (T_{max}) and the time to 25% recovery (duration 25%) of ST or T1 (first response in the TOF) were recorded. Further increments of the relaxant were administered at 25% recovery of ST or T1 if required. Halothane was discontinued immediately following administration of neostigmine.

Tracheal intubation was attempted 5 min after the administration of doxacurium. If unsuccessful it was tried again at the time of maximum block. The intubating conditions were graded as excellent, satisfactory, fair or poor according to a modification of the method of Lund and Stovner [11] and used previously by the authors [12, 13]. Excellent and satisfactory grades were considered clinically acceptable.

Neuromuscular monitoring was changed to the TOF mode in all 40 patients at the end of surgery before antagonism of block with neostigmine $50 \mu\text{g.kg}^{-1}$ and glycopyrronium $10 \mu\text{g.kg}^{-1}$. The height of the T1 and the TOF ratio were recorded before and after administration of the reversal mixture. The time taken to attain a TOF ratio of 70% and/or T1 of 90% or more were also recorded. Further smaller doses of neostigmine were administered if necessary.

Heart rate and indirect arterial blood pressure were recorded at 2, 5 and 10 min after relaxant administration.

The results were subjected to analysis of variance followed by Bonferroni testing if indicated.

Results

The mean (SD) age and weight of 10 patients in the first part (dose-response) of the study was 36 (11.2) years and 70 (14.5) kg respectively. Estimation of potency after construction of the cumulative dose-response curve (Fig. 1) showed the ED_{50} to be $15.75 \mu\text{g.kg}^{-1}$ (95% confidence limits 14.25–17.25) and ED_{95} to be $33.24 \mu\text{g.kg}^{-1}$ (95% confidence limits 27.40–39.08). Accordingly, a dose of $33 \mu\text{g.kg}^{-1}$ was administered to patients in the second part of the study.

The four groups in the second part of the study were not significantly different in their age, weight, height or sex distribution (Table 1). The lag time, T_{max} and the time to 25% recovery (duration 25) of ST or T1 are shown in Table 2. There was a wide variation among the values within each group. Patients developed an average block of greater than 90% and the lag time averaged more than one minute in all groups. Although maximum block occurred faster in those being given halothane and/or monitored using the TOF mode of stimulation, the differences were not significant (4.7 min in the TOF-halothane group, 8.5 min in the ST-fentanyl group). The duration to 25% recovery averaged from 52 to 72 min within the four groups but the differences within the groups were not significant.

The intubating conditions were excellent or satisfactory in all but two patients at 5 min; these, who were both in the single twitch-fentanyl group, were subsequently intubated uneventfully at the time of maximum block. The degree of block at the time of intubation ranged from 78 to 98% among the groups.

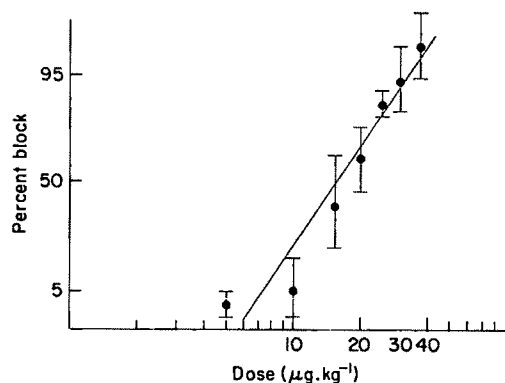


Fig. 1. Cumulative log dose-response curve for doxacurium. The points represent the degree of block produced by each successive increment and the bars represent this standard deviation.

Table 2. Onset and duration of clinical relaxation after administration of doxacurium $33 \mu\text{g.kg}^{-1}$ during halothane or fentanyl anaesthesia and after ST or TOF stimulation. Values are expressed as mean (SD).

	ST/Fentanyl (n = 9)	TOF/Fentanyl (n = 10)	ST/Halothane (n = 9)	TOF/Halothane (n = 9)
Lag time; min	1.6 (0.5)	1.6 (0.4)	1.3 (0.5)	1.2 (0.3)
Maximum block; %	92 (17)	97 (5)	98 (4)	100
T_{max} ; min	8.5 (4.6)	6.1 (1.9)	6.7 (1.8)	4.7 (1.3)
Duration 25; min	65 (22.8)	52.4 (21.7)	70 (33.4)	72 (21.0)

ST = single twitch; TOF = train-of-four; T_{max} = time to maximum block; Duration 25 = time to 25% recovery of ST or T1 in TOF (duration of clinical relaxation).

Table 3. Reversal data after administration of neostigmine $50 \mu\text{g.kg}^{-1}$. Values are expressed as mean (SD).

	ST/Fentanyl	TOF/Fentanyl	ST/Halothane	TOF/Halothane
T1; %, before neostigmine	46 (16.5)	39 (7.1)	41 (19.8)	34.8 (16.7)
TOF ratio before neostigmine	0.32 (0.13)	0.19 (0.1)	0.19 (0.09)	0.19 (0.07)
	(8)	(8)	(8)	(7)
Onset of effect; min	0.7 (0.4)	0.9 (0.3)	0.8 (0.5)	0.7 (0.2)
		(9)	(7)	(8)
Time to TOF ratio of 0.7; min	6.3 (5.5)	11.1 (10.5)	7.7 (5.0)	5.3 (2.4)
			(9)	(9)

ST = single twitch; TOF = train-of-four; n = 10 except where shown in parenthesis.

The data relating to antagonism are given in Table 3. The mean (SD) values of height of ST or T1 of TOF at the time of administering neostigmine varied from 35 (16.7)% in the TOF-halothane group to 46 (16.5)% in the ST-fentanyl group. There was no difference between the heights of ST or T1 or the TOF ratios in various groups. In three patients residual neuromuscular block had to be antagonised at a T1 of 5% as the action of muscle relaxant appeared to be very prolonged and circumstances did not permit us to wait for T1 to recover to 25%. Nine patients did not show a TOF response at the time of antagonism of neuromuscular block.

The mean (SD) times taken to achieve a satisfactory antagonism among the groups varied from 5.3 (2.4) to 11.1 (10.5) min, but with no statistically significant difference. Two patients in the halothane group did not attain a TOF of 70% even after the second dose of neostigmine and one of these patients did not attain a TOF of 70% even with a third dose of neostigmine and had to be ventilated in the recovery ward for about half an hour. All other patients satisfied the clinical criteria of extubation. None of the patients, including the one requiring ventilation, had any problems in the postoperative period and all made an uneventful recovery.

The haemodynamic data are shown in Table 4. There were no changes in heart rate and arterial pressure following administration of doxacurium nor was there any significant difference in haemodynamic parameters between the groups. There was no evidence of any histamine liberation or bradycardia (persistent heart rate of less than 50 min^{-1}) in any patient.

Discussion

The present study was designed to assess the potency and the time course of action of doxacurium and to assess the influence of different modes of stimulation on it. Estimation of potency showed the ED_{95} to be approximately $33 \mu\text{g.kg}^{-1}$ using the cumulative dose method. This is similar to that ($30 \mu\text{g.kg}^{-1}$) reported by Basta *et al.* using a single dose technique [1]. These findings support the view that cumulative and single dose techniques yield similar estimates of potency for longer-acting relaxants such as tubocurarine and pancuronium [8, 14]. Our estimated ED_{50} and ED_{95} are slightly higher than those obtained by Katz and colleagues [15] but this could be due to the use of TOF

Table 4. Heart rate (min^{-1}) and indirect arterial blood pressure (mmHg) after doxacurium administration. Values are expressed as mean (SD).

	Fentanyl anaesthesia				Halothane anaesthesia			
	SP	DP	MAP	HR	SP	DP	MAP	HR
Pre-induction	127 (19)	78 (11)	94 (10)	78 (12)	130 (19)	79 (15)	96 (16)	76 (13)
Pre-relaxation	113 (26)	64 (16)	78 (18)	71 (11)	114 (20)	66 (15)	83 (17)	63 (10)
+ 2 min	109 (23)	62 (13)	74 (16)	71 (10)	109 (15)	64 (13)	79 (14)	63 (10)
+ 5 min	110 (21)	62 (14)	74 (16)	71 (15)	111 (15)	65 (16)	82 (14)	67 (13)
+ 10 min	120 (26)	70 (18)	83 (21)	73 (12)	119 (23)	70 (15)	87 (18)	69 (9)

SP = systolic arterial blood pressure, DP = diastolic arterial blood pressure, MAP = mean arterial blood pressure, HR = heart rate.

mode of stimulation employed by them. Our study confirms doxacurium to be the most potent non-depolarising relaxant currently available, being approximately twice as potent as pancuronium (ED_{95} 72 $\mu\text{g.kg}^{-1}$) and about one and a half times as potent as pipecuronium (ED_{95} 45 $\mu\text{g.kg}^{-1}$) [9].

The onset of action of doxacurium in a dose of 1 ED_{95} is relatively slow, ranging from 3.4 to 13.1 min, with considerable interpatient variability similar to that reported in other studies [1, 2, 16]. The mode of stimulation did not make any significant difference to the onset time. This is in contrast to observation of a more rapid onset with the use of TOF mode of stimulation with intermediate acting agents atracurium and vecuronium [5, 6]. Apparently, the mode of stimulation probably makes no difference to the onset of action of long-acting relaxants.

The duration of clinical relaxation (duration 25) also showed very marked interpatient variability (range 31 to 103 min) although the mean values among the groups were not significantly different. This was irrespective of the use of halothane or the mode of stimulation. Katz and co-workers in a previous study showed similar results regarding the use of halothane [15]. Doxacurium thus appears to be an agent with a long duration of action, longer than that of both pancuronium and pipecuronium [17].

The intubating conditions were acceptable in 95% of patients when assessed at 5 min after administration of doxacurium. The conditions were not assessed before this, but with such a slow onset of action it is likely that these would not be good. In any case doxacurium would appear unlikely to be suitable for earlier intubation unless used in a large dose with a consequently prolonged action.

Reversal of residual neuromuscular block with neostigmine (50 $\mu\text{g.kg}^{-1}$) and glycopyrronium (10 $\mu\text{g.kg}^{-1}$) was satisfactory in 35 out of 40 patients when this was carried out at 25% recovery of ST or T1. In two patients, in whom T1 was only 5% at the time of administering neostigmine, recovery was delayed and did not reach a TOF ratio of 70% even with a second dose of neostigmine. Three other patients required a second dose of neostigmine (25 $\mu\text{g.kg}^{-1}$) even though the ST or T1 was greater than 25% of control at the time of administration of neostigmine. This again shows the marked individual variability in response to the doxacurium. In spite of these variations in response, all patients appeared to have appropriate antagonism of block clinically with good grip strength and sustained 5 s head lift when tested at 20 and 40 min in the recovery room.

Although not specifically designed to assess cardiovascular effects, the present study showed doxacurium to be free from any significant effects on heart rate and arterial blood pressure. However, detailed cardiovascular studies have also shown no cardiovascular effects [3, 4]. In this context the agent is similar to pipecuronium [18].

In conclusion, doxacurium is a potent but relatively slow and long-acting non-depolarising relaxant, with good cardiovascular stability. There is marked interpatient variability in its neuromuscular effects. When 25% or greater twitch height recovery was present, residual neuromuscular block could be satisfactorily antagonised with the neostigmine/glycopyrronium mixture. Neither the mode of stimulation nor the technique of anaesthesia seem to have any significant effect on the usually measured clinical variables.

Acknowledgments

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Ventilatory effects of subarachnoid fentanyl in the elderly

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Summary

Twenty-eight elderly patients scheduled for urological surgery were randomly assigned to receive, in a double-blind study, subarachnoid hyperbaric bupivacaine 15 mg with 50 µg (group A, $n = 7$), 25 µg (group B, $n = 7$), or 12.5 µg (group C, $n = 7$) of fentanyl or 1 ml of saline (group D, $n = 7$) in a total volume of 4 ml. The pattern of breathing and the ventilatory response to CO_2 were studied before and 90, 150 and 480 min after the subarachnoid injection. In group A, mild pruritus and sedation occurred in five patients, while nausea, vomiting and periodic breathing occurred in two. In group B, mild pruritus and sedation were observed in four patients, while nausea and vomiting occurred in two. No significant differences in minute ventilation, respiratory drive and respiratory timing were observed between the groups. Patients receiving fentanyl 50 µg showed a percentual change from baseline values as function of time (slope $\dot{V}_E/PE'CO_2$) significantly below baseline at 90 and 150 min ($p < 0.05$). However, the baseline values in this group reverted after 480 min. No side effects were observed in groups C or D. It is concluded that subarachnoid fentanyl 50 µg can cause an early respiratory depression and its use as a postoperative analgesic should be avoided in the elderly.

Key words

Anaesthetic techniques, regional; spinal.
Analgesics; fentanyl.

Previous studies have shown that opioids binding to sites in the spinal cord can produce analgesia in laboratory animals [1] and humans [2]. It has also been observed that the intrathecal or epidural administration of opioids may be accompanied by a number of side effects [3]. The most undesirable side effect of spinal narcotics is moderate to severe respiratory depression, which may occur early due to drug-bearing blood reaching the brain (early effect) [4] or late due to cephalad drug migration in cerebrospinal fluid (CSF) (up to 12 h after the initial administration [5, 6]. Several retrospective studies attribute delayed respiratory depression to upward cephalad drift of opioid in the CSF [7–11].

The majority of reports on lipophilic opioid administration make mention of brief periods of early respiratory depression [7, 8, 11]. A single dose of epidural fentanyl, while it does not appear to give rise to delayed respiratory depression [12], produces changes in respiratory frequency as well as in end-tidal CO_2 within minutes of administration [13, 14] which are not clinically detectable. No respiratory changes are discernable during continuous

infusion of epidural fentanyl, but a mild, significant, ventilatory depression was evident during the CO_2 stimulation test [15]. Subarachnoid fentanyl has not been used extensively, but recently it has been shown that its use in selective doses improves both intra and postoperative analgesia [16, 17]. Side effects were mild, mostly pruritus. No change in respiratory rate, even in high doses [17] or clinical respiratory depression has been reported in elderly patients [18].

The present randomised dose-response, double-blind study, was undertaken to evaluate the ventilatory effects of different doses of subarachnoid fentanyl in elderly patients.

Methods

Twenty-eight male patients scheduled for elective trans-urethral or suprapubic prostatectomy, and from whom informed consent was obtained, were studied. The study protocol was approved by the ethics committee. All the patients fasted overnight, did not take caffeine or alcohol-containing beverages, and were not premedicated before

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Table 1. Age, weight and height of patients studied, values given as mean (SD).

Group	A	B	C	D
Age; years	71 (6.8)	70.2 (5.4)	68.4 (4.9)	69 (6.1)
Weight; kg	66.8 (7.9)	68.4 (6.1)	70.3 (1.2)	70 (7.5)
Height; cm	170.1 (3.7)	165.9 (7.4)	169.3 (4.4)	169.5 (3.3)

Groups: A = fentanyl 50 μ g, B = fentanyl 25 μ g, C = fentanyl 12.5 μ g, D = saline.

surgery. They did not smoke before the study for at least a week. An intravenous infusion of Ringer's lactate solution 500 ml was given before the start of the block. The subarachnoid block was performed with a standard technique using a 25 gauge needle at the L₂₋₃ or L₃₋₄ interspace, with the patient in the sitting position.

The patients were randomly allocated to receive subarachnoid hyperbaric bupivacaine 15 mg with the addition of 50, 25 or 12.5 μ g fentanyl (groups A, B and C respectively) or 1 ml of saline (control, group D) for a total volume of 4 ml. The specific gravity of the solutions injected was 1030 ± 2 for the fentanyl groups and 1032 ± 1 for the controls. The pH was 6.4 ± 0.005 for fentanyl groups, and 5.92 ± 0.007 for controls.

After the block, all the patients were maintained in a 30° head-up position. Intra-operative hypotension was considered to be present whenever systolic blood pressure decreased more than 30 mmHg or to less than 100 mmHg. All patients received intravenous prophylactic ephedrine 5 mg and further doses were administered if hypotension occurred. The cephalad level of spinal anaesthesia was assessed every 5 min by pinprick until the level became unchanged on two successive tests.

Ventilatory measurements were made in all patients while resting in a supine 30° head-up position, before (baseline value) and 90, 150 and 480 min after subarachnoid injection. The following parameters were evaluated: respiratory rate (RR), minute ventilation ($\dot{V}_E$), respiratory drive, respiratory timing and the ventilatory response to CO₂. The ventilatory response to CO₂ was

measured while the patients were breathing room air and using Read's rebreathing method [19]. The subjects rebreathed CO₂ through a mouthpiece connected to a bell-ventilated Godart spirometer (Pulmotest). End-tidal CO₂ (P_{E'}CO₂) was measured with an IL200 capnograph, calibrated before and after each measurement with 5 and 7% CO₂ in O₂ from calibrated tanks, which were verified to be accurate within 1% of the assigned value using Micro Scholander analysis.

During air-breathing, a steady state was obtained after 3 to 7 min, with stabilisation of P_{E'}CO₂ values. All the resting values reported are the means of 10 breaths obtained at this time. Moreover, in subjects breathing from the spirometer, initially filled with 7% CO₂ in O₂, measurements from the first 30–40 s were discarded until a linear rising P_{E'}CO₂ phase was observed at approximately 50 mmHg. For each of the subsequent 3–4 min, P_{E'}CO₂ and $\dot{V}_E$ readings were calculated at 30 s intervals. Rebreathing was continued until P_{E'}CO₂ increased to 70 mmHg. After converting ventilatory variables to body temperature and ambient pressure saturated, the slope of ventilatory response to CO₂ was determined by least squares linear regression of $\dot{V}_E$ on P_{E'}CO₂. All responses were linear, with the correlation coefficients (r) ranging between 0.91 and 0.98. Respiratory drive and respiratory timing were calculated from the tidal volume trace by maximum inspiratory flow (V_t/T_i) and inspiratory time–total breathing time ratio (T_i/T_{tot}) respectively.

Heart rate, arterial blood pressure and respiratory rate were noted every 2 h. All patients were monitored by pulse

Table 2. Ventilatory variables (SD).

	Min	Group A	Group B	Group C	Group D
RR; breath.min ⁻¹	Baseline	18 (3)	16 (2)	18 (2)	14 (2)
	90	20 (3)	18 (1)	19 (1)	16 (2)
	150	20 (2.8)	20 (2)	18 (2)	19 (1)
	480	19 (1)	18 (3)	16 (2)	17 (3)
P _{E'} CO ₂ ; kPa	Baseline	4.0 (0.26)	3.8 (0.30)	4.0 (0.24)	4.0 (0.33)
	90	3.8 (0.25)	4.0 (0.28)	3.6 (0.40)	3.5 (0.28)
	150	4.0 (0.30)	4.0 (0.22)	3.7 (0.20)	4.0 (0.16)
	480	3.8 (0.20)	3.7 (0.28)	4.1 (0.26)	4.0 (0.21)
$\dot{V}_E$; l.min ⁻¹	Baseline	10.5 (4.8)	8.9 (4.1)	9.1 (3.1)	7.2 (2.5)
	90	8.3 (3.5)	10.1 (3.6)	9.4 (2.2)	9.4 (3.2)
	150	9.5 (3.2)	7.9 (5.1)	10.2 (3.1)	9.2 (2)
	480	10.2 (2.6)	8.3 (1.4)	8.1 (1.1)	10.4 (2.4)
V _t /T _i ; l.min ⁻¹	Baseline	487 (160)	426 (127)	438 (112)	377 (76)
	90	413 (185)	455 (101)	396 (98)	421 (106)
	150	389 (188)	377 (97)	388 (117)	409 (113)
	480	423 (102)	438 (113)	399 (96)	411 (89)
T _i /T _{tot}	Baseline	0.350 (0.070)	0.340 (0.450)	0.350 (0.062)	0.320 (0.050)
	90	0.340 (0.046)	0.360 (0.071)	0.330 (0.059)	0.370 (0.058)
	150	0.350 (0.042)	0.350 (0.058)	0.350 (0.061)	0.340 (0.044)
	480	0.360 (0.039)	0.350 (0.071)	0.340 (0.045)	0.340 (0.061)

Groups: A = fentanyl 50 μ g, B = fentanyl 25 μ g, C = fentanyl 12.5 μ g, D = saline.

RR, respiratory rate; $\dot{V}_E$, minute ventilation; P_{E'}CO₂ = end-tidal tension of CO₂; V_t/T_i, respiratory drive; T_i/T_{tot}, respiratory timing.

Table 3. Nonrespiratory side effects (number of patients).

Group	A	B	C	D
Nausea	2 (28.5%)*	2 (28.5%)	2 (28.5%)	1 (14.3%)
Vomiting	2 (28.5%)*	2 (28.5%)	—	—
Itching	5 (71.4%)	4 (57.1%)	—	—
Drowsiness	5 (71.4%)	4 (57.1%)	—	—

Groups: A = fentanyl 50 µg, B = fentanyl 25 µg, C = fentanyl 12.5 µg, D = saline.

*Nausea and vomiting occurred in the same two patients at the time of periodic breathing.

oximetry and transcutaneous O_2/CO_2 continuously intra-operatively and for 120 min postoperatively. Postoperative analgesia was measured as time to the first patient's request for analgesic medication. All patients were observed and asked about the presence or absence of nausea, vomiting, pruritus and drowsiness.

Statistical analysis was performed by analysis of variance and Student's Newman Keultz test when appropriate. A *p* value less than 0.05 was considered significant.

Results

There were no significant differences between the groups in respect of age, weight and height (Table 1). No significant differences in vital signs, O_2 saturation and transcutaneous O_2/CO_2 were observed, and there was no apnoea in groups B, C or D. No significant changes in RR, $\dot{V}_E$, V_t/Ti , Ti/T_{tot} and $PE'CO_2$ values were observed in any of the groups (Table 2).

In groups B, C and D the slope $\dot{V}_E/PE'CO_2$ did not change significantly from baseline values. In group A the slope $\dot{V}_E/PE'CO_2$ was significantly below baseline values at 90 and 150 min (*p* < 0.05) (Fig. 1). Apnoea and periodic breathing were observed in two patients who received subarachnoid fentanyl 50 µg.

Mild pruritus and sedation occurred in five patients in group A (71.4%) and four in group B (57.1%). Nausea and vomiting occurred in the same two group A patients (28%) who had periodic breathing and was observed in two patients (28%) in group B (Table 3). Postoperative analgesia is reported in Table 4. Patients who received the 50 and the 25 µg dose had longer postoperative analgesia than the other two groups.

Table 4. Postoperative analgesia, values given as mean (SD).

Group	A	B	C	D
Time (min)	278 (38)	220 (41)	141 (27)	126 (32)

Groups: A = fentanyl 50 µg, B = fentanyl 25 µg, C = fentanyl 12.5 µg, D = saline.

Discussion

The major advantage of pain relief by spinal opioids is the absence of motor and sympathetic blockade and of postural hypotension. The benefits to the patient include adequate postoperative analgesia, better pulmonary function and earlier ambulation [20].

However, the use of spinal opioids for postoperative analgesia is not without side effects. They can cause nausea, vomiting, urinary retention, pruritus and respiratory depression. All opioid agonists produce dose-related respiratory depression by decreasing the sensitivity of medullary respiratory centres to hypercapnia. Therapeutic doses of opioids may slow respiratory rate and produce irregular or periodic breathing [21] that may be followed by sudden apnoea, especially if other risk factors such as concomitant use of central nervous system depressing drugs or ageing are present [4]. These effects were observed more often with morphine and less frequently after fentanyl or its derivatives [22]. Lipophilic drugs bind rapidly to the nervous tissue, therefore, if injected in the lumbar region, their ability to reach the medullary respiratory centres is dose dependent [8, 9, 23]. Respiratory rate is an insensitive measure for monitoring the respiratory depression. In fact, the response to the CO_2 stimulation may be decreased in the face of a normal respiratory rate and apparently normal state of consciousness. This finding suggests that subarachnoid fentanyl may produce early respiratory depression if used in high doses [2] in spite of a normal range of the respiratory rate.

It has been reported that the effect of spinal opioids is modulated by a number of factors including pKa, lipid solubility, molecular weight [20], baricity of the solution, concentration of drugs in CSF [3], age and posture of the patient. In this study, the sitting position and the use of an hyperbaric solution did not prevent respiratory depression. According to the pharmacokinetics of opioids in elderly

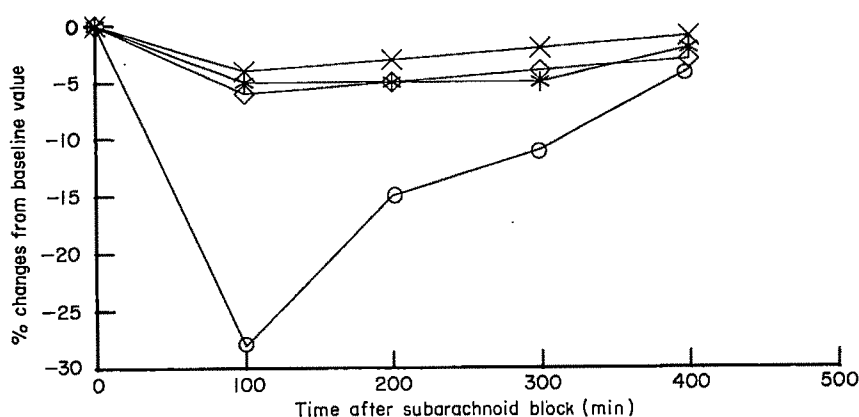


Fig. 1. Percent changes in the $\dot{V}_E/PE'CO_2$ slope from baseline value as a function of time (min) following the subarachnoid administration of fentanyl or saline. Group A (○) fentanyl 50 µg; group B (◇) fentanyl 25 µg; group C (*) fentanyl 12.5 µg; group D (×) saline.

patients, this depression may be linked to a high concentration of fentanyl in the brain, probably partly caused by a shift of a large portion of the drug into the plasma, and partly because of a decrease in blood flow to organs in the vessel-rich group [24], shifting the drug to vital organs such as the brain [25]. In our cases, a higher peak CSF concentration of fentanyl could have produced an early and prolonged depression of ventilation. This is suggested by the drowsiness of the patients and the connection between nausea and vomiting and the occurrence of periodic breathing in the same patients. In effect it was demonstrated that the fentanyl in the lumbar CSF, in high dose, undergoes cephalad migration because of passive CSF flow, resulting in measurable fentanyl concentration in cervical CSF samples [23]. Fentanyl enters the brain rapidly [26] and the half-time of the terminal elimination phase in CSF has been reported to be 178 (SD 8) min [27].

Opioid effects that are mediated by receptors located exclusively in the brain, may require much higher concentration of the drug and thus will become apparent only at considerably higher doses [2]. In addition, there are a number of factors that may have affected the early rostral spread of fentanyl in our patients. Elderly patients are more susceptible to respiratory depression by opioids [10]. Opioid accumulation changes with ageing [28], probably as a result of progressive decrease in clearance of opioid associated with increased age [29]. The mean age of this study population was 70 years. During spinal opioid administration, older patients can maintain a normal respiratory frequency, but with severe decrease in their tidal volume and often severe hypercarbia. Hypoxia may develop if patients do not receive supplemental oxygen [30]. The difference in the pattern of ventilation in this study group resulted from age-related changes in neuromuscular, rather than CNS function [31]. Our patients did not receive any O₂ supplement, in order not to interfere with the study of ventilation.

Ageing changes the anatomical configuration of the lumbar and thoracic spine and possibly reduces the volume of CSF. These alterations may contribute to increased spread of the solution injected in the subarachnoid space [32].

In conclusion, the early onset of respiratory depression, due to a single dose of fentanyl, results from its rapid entry into the CNS associated with the portion of the drug which arrives at the brain via the circulatory system. The intensity and duration of respiratory depression may be correlated to the concentration of the drug in the CSF. Patients receiving subarachnoid fentanyl 50 µg had prolonged post-operative analgesia and respiratory depression, as shown by the depressed response to the CO₂ stimulation test. Subarachnoid fentanyl 25 µg seems to be the only dose which gives significant analgesic effect without respiratory depression in the older patient.

The potential risk of respiratory depression must always be considered when intraspinal narcotics are used [33] and careful monitoring, especially in the elderly, is a key to their safe use as postoperative analgesics.

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Circulatory responses during electroconvulsive therapy

The comparative effects of placebo, esmolol and nitroglycerin

D. O'FLAHERTY, M. M. HUSAIN, M. MOORE, T. R. WOLFF, S. SILLS AND A. H. GIESECKE

Summary

Electroconvulsive therapy is usually accompanied by activation of the autonomic nervous system, which may be harmful in patients with cerebrovascular or ischaemic heart disease. We have compared this haemodynamic response in a series of 82 electroconvulsive treatments randomly assigned to receive either nitroglycerin $3 \mu\text{g.kg}^{-1}$, esmolol 2 mg.kg^{-1} or placebo. These drugs were given shortly after the suxamethonium and 2 min before the electroconvulsive therapy in all cases. Heart rate was significantly lower with esmolol 1 min after therapy as was blood pressure (systolic and diastolic). The pulse rate was higher following nitroglycerin than placebo, which in turn was higher than esmolol. The three groups did not differ with regard to seizure duration. The results demonstrate that esmolol is more effective than nitroglycerin in controlling the haemodynamic response to electroconvulsive therapy. With recent emphasis on stabilisation of heart rate in preference to blood pressure in at-risk cardiac patients, our study suggests that, in the doses selected, esmolol is preferred to nitroglycerin to control the heart rate response to electroconvulsive therapy.

Key words

Anaesthesia; electroconvulsive therapy.

Pharmacology; esmolol, nitroglycerin.

Electroconvulsive therapy (ECT) is a widely used treatment for depression and is accompanied by activation of the autonomic nervous system [1]. The potential for life-threatening complications associated with ECT in patients with coronary artery disease, systemic arterial hypertension, aneurysmal vascular disease, and decreased intracranial compliance are well known [2]. The circulatory disturbances consist of alterations in heart rate and elevations in systemic arterial blood pressure, which occasionally lead to myocardial ischaemia, heart failure and cerebrovascular catastrophes. These changes stem from reflex autonomic discharge resulting from central stimulation, associated with increased plasma catecholamine concentrations. The ECT-induced transient hypertension and tachycardia may be harmful to those patients with cerebrovascular and ischaemic heart disease [3, 4].

Recommended pretreatment regimens to block the ECT-induced cardiovascular responses have included lignocaine, vasodilators, adrenergic blockers and narcotics. The new adrenoceptor antagonist, esmolol, is an attractive option because of its cardioselective beta receptor-blocking

properties, and its short duration of action [5], but it has not been systematically compared with vasodilators with regard to relative potency in obtunding ECT-induced responses. This study was designed to compare the circulatory effects of esmolol, nitroglycerin and placebo on the haemodynamic response and seizure duration induced by ECT.

Methods

After institutional approval and written informed consent, 10 patients undergoing a total of 82 ECT treatments were enrolled in a prospective randomised study. Exclusion criteria are shown in Table 1. No patient was receiving antihypertensive therapy at the time of hospital admission or during the study and all patients had their psychotropic medications e.g. antidepressants or benzodiazepines weaned before receiving ECT. Patients were monitored with a precordial stethoscope and pulse oximeter. In addition, records were made of heart rate, indirect blood pressure, and five-lead electrocardiogram (ECG). One

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Table 1. Exclusion criteria.

Diabetes
Pregnancy
Less than 21 years old
Ischaemic heart disease
Hypertension
Bronchospasm or bronchial asthma
Drug allergy or idiosyncrasy to beta-adrenergic drugs
Experimental drugs within 2 weeks
Patients receiving beta-blockers or calcium channel blockers

channel electroencephalogram (EEG) was obtained using frontal leads. An intravenous cannula was inserted. After baseline blood pressure and heart rate measurements were made, patients breathed oxygen through a mask for 5 min.

Doses of the study drugs were chosen based on the literature and on previous clinical experience [6–8]. The esmolol dose was based on a previous study from this department examining the efficacy of different esmolol dosages in attenuating the cardiovascular responses to tracheal extubation [9]. The nitroglycerin dose used was based on haemodynamic data obtained during a small pilot study on patients undergoing ECT. The patients were studied during sequential ECT treatments administered three times per week. Using a cross-over design, each patient received each study drug on three occasions during a total of nine consecutive treatment sessions. The order of the treatments was determined by a random table prepared before the study of the first patient.

Each patient received one to three ECT treatments before entering the study. This allowed determination of the most appropriate doses of methohexitone and suxamethonium for anaesthetic induction as these doses can vary between patients. Once doses of methohexitone and suxamethonium, sufficient to produce anaesthesia and paralysis without prolonged awakening time, had been established, these doses were used throughout the study, and ranged from 0.9 to 1.5 mg.kg⁻¹ (1.1 (0.06) mg.kg⁻¹, mean (SEM)) and from 0.5 to 1.2 mg.kg⁻¹ (1.0 (0.06) mg.kg⁻¹), respectively.

Five minutes after administration of 0.2 mg glycopyrronium, anaesthesia was induced with methohexitone. When the patient became unresponsive, ventilation was assisted via a mask. At that time a blood pressure cuff above the right ankle was inflated to 300 mmHg to isolate the foot. The patient then received suxamethonium. The study drug, esmolol 2 mg.kg⁻¹, nitroglycerin 3 µg.kg⁻¹ or placebo (sodium chloride 0.9% 10 ml) was then given intravenously 2 min before ECT. The patient's lungs were hyperventilated with oxygen for 30s prior to application of the electrical stimulus. Right unilateral stimulus, using the MECTA SR1 ECT machine, was started at a low energy and gradually increased to assess the seizure threshold during the first control treatment. Once the seizure threshold was estimated, subsequent treatments were maintained at energy levels 100% above this. If no seizures occurred after the initial stimulus, data from this treatment were excluded from the study. If the patient's condition did not improve over the course of several treatments, bilateral ECT, if considered appropriate, was administered.

Heart rate, systolic and diastolic blood pressure were recorded prior to glycopyrronium administration, before

induction, prior to ECT treatment, and each min for 15 min after ECT. Seizure duration, both EEG and motor as observed from the isolated foot were also recorded. The blood pressure, heart rate and EEG tracings were analysed later by an individual who was blinded to the treatment regimens. Data were analysed by repeated measure analysis of variance and $p < 0.05$ was considered statistically significant.

Results

In the 10 patients studied, the median (SD) age was 44 (12) years (range 26–61) and mean weight 66 (12) kg (range 55–88). The male to female ratio was 4:6.

Control studies

During control studies, ECT significantly increased arterial blood pressure and heart rate in every patient. Haemodynamic response to ECT varied considerably between patients ($p < 0.001$) with mean increases in blood pressure of 150% ($p < 0.05$) compared with baseline and in heart rate of 160% ($p < 0.01$) expressed as a percentage of the pre-glycopyrronium control value. All initial (control) blood pressure and heart rate values were within the normal range and subsequent blood pressure and heart rate values are expressed as absolute values.

Haemodynamic variables

No differences were found between treatment groups in blood pressure or heart rate at the initial measurement, 2 min after glycopyrronium administration, or immediately following anaesthetic induction. Significant increases occurred in heart rate with anaesthetic induction, with no significant differences between the study groups. Significant differences were noted, however, in both diastolic and systolic blood pressure and heart rate between the study regimens immediately after ECT stimulation was applied. Smaller increases in diastolic and systolic blood pressure were noted after esmolol pretreatment than after nitroglycerin or saline pretreatment ($p < 0.05$) (Figs 1 and 2). Esmolol significantly attenuated the tachycardia seen following saline and nitroglycerin treatments ($p < 0.01$) at 1, 2 and 3 min after ECT stimulation (Fig. 3). The pulse rate was higher following nitroglycerin than placebo, which in turn was higher than esmolol. Differences between the study regimens were significant only in the first minute following the electrical stimulus for blood pressure, and in the 1–3 min post-stimulation period for heart rate, after which the measured parameters quickly returned to control values.

During the present study, one clinically significant episode of sinus arrest was observed out of 82 treatments examined. This occurred immediately after ECT stimulation in a patient receiving the placebo treatment. The overall incidence of arrhythmias was 15% and was lowest in those patients receiving esmolol. However, this observation was not statistically significant. Nineteen (23%) of the ECT treatments involved the use of bilateral electrical stimulation. The haemodynamic responses did not differ either

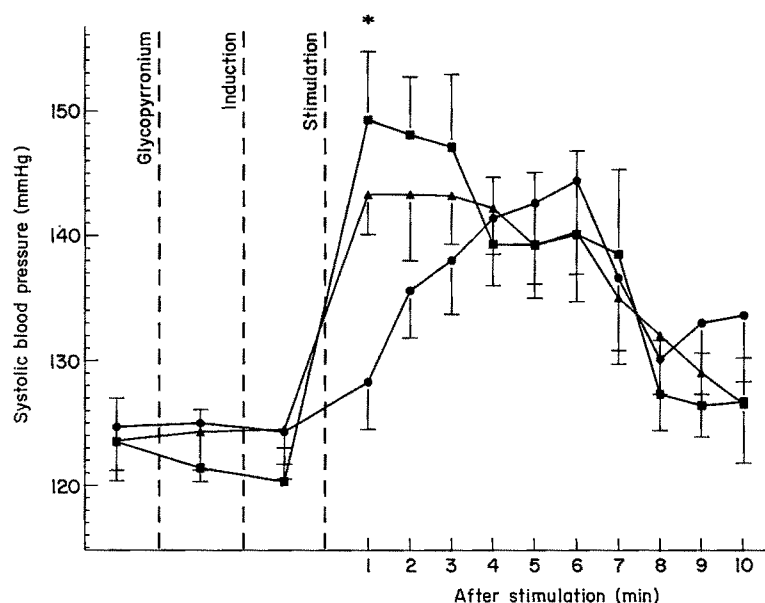


Fig. 1. Systolic blood pressure response to electroconvulsive therapy (* $p < 0.05$ between groups). ●, esmolol; ▲, nitroglycerin; ■, placebo.

during the modified or unmodified treatments in comparison with the circulatory responses to unilateral stimulation.

Seizure duration

Seizure duration, as measured by EEG, did not correlate with that observed for isolated limb myoclonic activity. In general, the electrical manifestations of the seizure were present for 15–30 s longer than motor seizures, and treatment groups were not significantly different with regard to seizure duration, as assessed either by motor activity or EEG.

Emergence

The heart rate response on awakening was attenuated, but not significantly, by the different treatment regimens in a manner similar to their circulatory effect immediately after seizure. Esmolol's effectiveness was, however, much less pronounced, and the heart rate response in esmolol-treated individuals was not significantly different from that of control patients. Heart rate was higher following nitroglycerin than placebo ($p = 0.077$ at 7 min), which in turn was higher than esmolol ($p = 0.08$) at 7–10 min after ECT stimulation, which coincided with emergence. The blood pressure response on awakening was attenuated but not

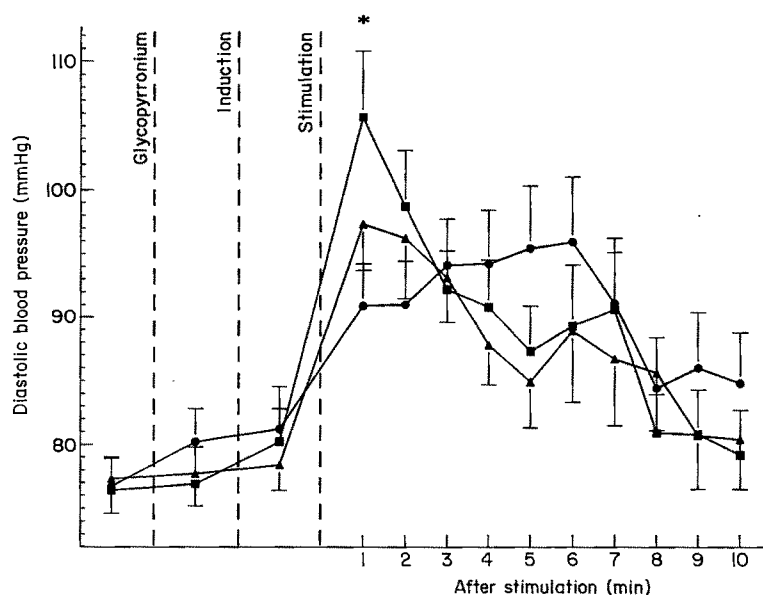


Fig. 2. Diastolic blood pressure response to electroconvulsive therapy (* $p < 0.05$). ●, esmolol; ▲, nitroglycerin; ■, placebo.



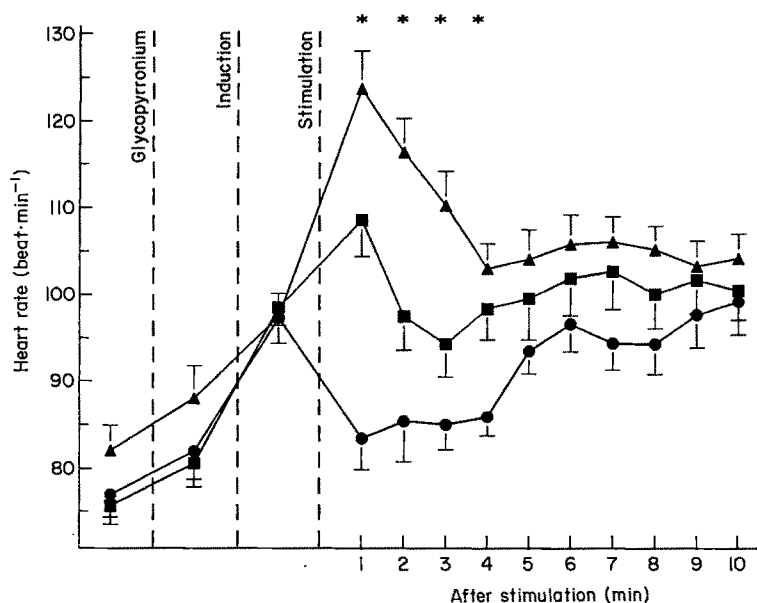


Fig. 3. Heart rate response to electroconvulsive therapy (* $p < 0.01$), ●, esmolol; ▲, nitroglycerin; ■, placebo.

significantly ($p = 0.85$ at 7 min) by the different treatment regimens in a reverse manner to their circulatory effect immediately after seizure.

Discussion

Interest in electroconvulsive therapy (ECT) for the treatment of patients with major affective disorders has recently experienced a renaissance. American studies report that 4% of psychiatric admissions, approximately 100 000 patients per year, are given electroconvulsive therapy. In the UK, approximately 200 000 treatments are administered each year. While these figures are a significant reduction compared with earlier decades, the majority of these patients respond favourably. Increased use of pharmacological agents, regulatory pressures and unwillingness to maintain the necessary staff and equipment for ECT, account for the early decline which has now abated and ECT usage is probably on the increase again [10]. ECT is used when the patient fails to respond to conventional psychopharmacological therapy or suffers severe side effects of the drugs [11].

Biophysiological changes associated with electroconvulsive therapy effect principally the cardiovascular and central nervous systems. The cardiovascular changes induced by ECT are a consequence of initial vagal stimulation followed by sympathetic stimulation, concluding with a mixture of combined activation and their compensatory mechanisms. The initial parasympathetic discharge occurring during the tonic phase of the induced seizure immediately after the application of the current produces a bradycardia or sometimes even asystole with a consequent fall in blood pressure lasting about 30 s. This is followed by sympathetic nervous system activation, which closely mirrors the clonic phase, with an increase in heart rate and blood pressure. The marked tachycardia arises initially from direct adrenergic outflow through paravertebral

sympathetic ganglia but is sustained by catecholamines released from the adrenal medulla [1, 4].

Although rare, cardiovascular complications are the main cause of death during ECT with a mortality rate of 0.03% of patients treated, and 0.0045% of individual ECT treatments [2, 4, 6]. This is higher than the often quoted overall anaesthetic mortality of 1:10 000 [12]. Morbidity and mortality during ECT is usually secondary to myocardial ischaemia or a cerebrovascular accident. Patients who are elderly, have chronic hypertension, or pre-existing cardiac disease are especially at risk for an adverse event occurring in the peri-ECT period [13–15].

Various therapeutic interventions are presently being used to dampen the significant cardiac variability experienced with the administration of ECT. Studies have shown the possible attenuation of ECT-induced hypertension and tachycardia by prior administration of intravenous or topical nitroglycerin, oral clonidine, intranasal nifedipine and adrenergic blockers [16–21]. Pretreatment with sodium nitroprusside, although successful in attenuating the hypertensive response, is often associated with persistent hypotension after ECT. Vasodilators and lignocaine provide an incomplete solution, controlling hypertension, but not affecting heart rate. Propranolol's effects outlast the stressful interval by several hours, and not infrequently include increases in bronchomotor tone. Although numerous agents are available, many have side effects or have durations of action longer than the short ECT stimulus itself.

The ideal drug for attenuating the hyperdynamic response of ECT would be convenient, easy to prepare and administer, rapid-acting, brief, nontoxic, and have minimal or no side effects [6]. Ideally, duration of action should correspond to the period of time the drug and/or technique is needed. We conclude from this study that esmolol is superior to nitroglycerin or placebo in attenuating these responses and possesses many of the qualities of the ideal theoretical agent for use during a short-lived

stress such as ECT. The duration of the brief adrenergic blockade with esmolol appears similar to the peak sympathetic stimulation after ECT. The cardiovascular attenuation with esmolol suggests a potential beneficial effect on myocardial oxygen balance [22]. By contrast, nitroglycerin further increased heart rate, an important determinant of myocardial oxygen consumption.

Esmolol is an ultrashort-acting beta-one selective blocker with a distribution half-life of 2 min and an elimination half-life of 9 min [5]. Administered by bolus or infusion, it has been shown to be effective in obtunding the haemodynamic effects of other peri-operative stresses [23–26]. We administered esmolol exactly 2 min before ECT shock since a bolus dose given 3 to 4 min before the stimulus may be too early [6]. While esmolol bolus is more convenient and easier to administer than the infusion method, a more brief control of heart rate also occurs. Repeat bolus doses or an infusion may be required post-ECT if further control of heart rate and blood pressure is desired.

While the administration of glycopyrronium made the analysis of the haemodynamic data more difficult, it was considered necessary to protect against the well documented vagal surge and sialorrhea after the application of stimulus. The dose was fixed at 0.2 mg and was given to all patients. Our data also suggest that the mode of electrical stimulation does not greatly influence the body's circulatory response to the induced seizure, whether or not a pharmacological modifier has been administered. However, as the number of treatments involving bilateral stimulation was small, this aspect of the study deserves further investigation, as the study of greater numbers of treatments may reveal more subtle haemodynamic response differences.

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Clinical use of a small soda lime canister in a low-flow to-and-fro system

G. G. LOCKWOOD, M. Y. KADIM, M. K. CHAKRABARTI AND J. G. WHITWAM

Summary

It may be possible to overcome the classical disadvantages of a to-and-fro system by the use of a small soda lime canister. We report our experience of such a system in a series of 13 adult patients requiring mechanical ventilation of the lungs during surgery. The system was found to be convenient to use and the charge of soda lime had sufficient absorptive capacity to last throughout the great majority of surgical cases. Using a fresh gas flow of 1 l.min^{-1} , the ratio of inspired isoflurane concentration to isoflurane vaporizer setting was found to be approximately one half after 10 min of anaesthesia. This ratio was maintained throughout the procedure, except for a few minutes following each change in vaporizer setting. A to-and-fro system using a small soda lime canister is a convenient alternative to the circle system for low-flow anaesthesia.

Key words

Anaesthetics, volatile; isoflurane.

Carbon dioxide; absorption.

Equipment; absorbers.

Low-flow anaesthetic systems are attractive for financial and environmental reasons. Attention has focused on the circle system rather than the to-and-fro. However, the circle system is not perfect: it is a cumbersome attachment to the anaesthetic machine requiring double corrugated tubing, and the two passive valves may stick when wet. A to-and-fro system, with a small, lightweight soda lime chamber close to the patient's head is a comparatively convenient system for clinical use. The small size of the canister limits the operation time unless it can be replaced conveniently but it may overcome the usual objections to the to-and-fro system: the inconvenience of a bulky canister close to the patient's head, and the increase in apparatus deadspace as the soda lime becomes exhausted. Detailed laboratory evaluation of the performance of such a soda lime canister has been reported recently [1], and we comment here on its use in clinical practice. We have also examined the predictability of inspired anaesthetic concentration relative to the vaporizer setting during low-flow anaesthesia, and we make some general observations and comments on low-flow anaesthesia.

Methods

The hospital Ethics Committee approved this study; the to-and-fro system had not been used in this hospital for many years. Thirteen patients of ASA grades 1, 2 or 3 gave their informed consent to take part (Table 1). Patients were due to undergo surgery expected to last more than 1 h, and the anaesthetist planned mechanical ventilation.

The patients were premedicated with papaveretum and hyoscine. Anaesthesia was induced with either thiopentone or propofol, and muscle relaxation was produced by a non-depolarising agent. The patients' lungs were ventilated with 100% oxygen until tracheal intubation and connection to the to-and-fro breathing system. Fresh gas (50:50 nitrous oxide and oxygen) was supplied to the system at 1 l.min^{-1} with an initial isoflurane concentration of 5%, and anaesthesia was controlled by adjusting the concentration of isoflurane in the fresh gas (C_{FGF}). Additional doses of intravenous anaesthetic were given as judged appropriate until inhalation anaesthesia was established. The tidal volume was set to 10 ml.kg^{-1} and ventilation was adjusted

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Table 1. Patient data.

Patient	Sex	Age (years)	Weight (kg)	Duration (min)	Asymptotic C_I/C_{FGF}	Time to inspired concentration (min)			Peak CO_2 (%)
						1% isoflurane	40% N_2O	0.5% CO_2	
1	M	54	80	137	0.50	2	7	81	1.6
2	F	45	68	115	0.60	2	12	—	0.3
3	F	36	63	15	0.50	1	4	—	0.3
4	F	32	50	76	0.50	1	10	—	0.4
5	F	30	64	67	0.50	2	14	—	0.2
6	M	41	62	56	0.65	3	18	—	0.2
7	M	66	93	130	0.50	2	4	94	0.9
8	M	32	81	56	0.55	3	15	45	0.5
9	F	36	54	116	0.50	1	11	98	0.5
10	M	70	76	142	0.60	2	8	—	0.3
11	M	22	*	**148	0.45	3	20	45	1.3
12	F	49	80	203	0.50	***12	***30	124	1.7
13	F	25	*	18	0.40	2	16	12	0.5
Mean		41	70	94	0.52	2.0	11.6	71	
SD		15	13	56	0.07	0.7	5.2	39	

*Data not available.

**The soda lime was changed after 135 min in case 11.

***FGF only $700 \text{ ml} \cdot \text{min}^{-1}$ for the first 58 min of anaesthesia in case 12. These data are excluded from the mean and SD.

by control of respiratory rate to maintain normocapnia. When the patients were stable, the respiratory rate was increased by 50% until the end-tidal carbon dioxide tension fell to 4 kPa. At this time the respiratory rate was halved until the patient became normocapnic, and then adjusted to maintain this state. At the end of the procedure, muscle paralysis was antagonised by neostigmine and atropine, and the fresh gas flow (FGF) was increased to facilitate isoflurane elimination.

The breathing system is shown in Figure 1. A paediatric soda lime canister, 5 cm diameter and 9 cm in length, was used and charged with $160 \text{ g} \times 8$ mesh soda lime (Durasorb MIE). A valveless ventilator [2] was connected to the soda lime canister via a length of 22 mm plastic corrugated tube of 2 l internal volume. This tube prevents mixing between the driving gas and anaesthetic gas [3]. The other end of the canister was attached to the patient's tracheal tube via an inlet port for the fresh gas, an Engstrom Edith catheter mount with integral heat and moisture exchanger (HME), and a connector which allowed continuous sampling and return of gas for analysis. The aspiration port of this connector had a catheter extending almost to the tracheal end of the tracheal tube, with numerous fenestrations at its tip. Gas was returned to the system at the mouth end of the tracheal tube. Gas analysis was performed by a Datex Capnomac [4] calibrated using 'QuickCal' calibration gas before each anaesthetic. Every 10 s, data output from the

Capnomac were collected by an IBM XT computer, and four analogue outputs from the Capnomac (oxygen, carbon dioxide, nitrous oxide and isoflurane concentrations) were recorded continuously on a pen recorder (Lectromed Multitrace 4).

Fresh gas was supplied by an Ohmeda Excel 410 anaesthetic machine via a 1.5 m, 3 mm internal diameter tube. The nitrous oxide flow meter on this machine is not calibrated below a flow of $1 \text{ l} \cdot \text{min}^{-1}$, and so a 50:50 oxygen: nitrous oxide mixture was obtained by setting the oxygen flow to $500 \text{ ml} \cdot \text{min}^{-1}$ and adjusting the nitrous oxide flow to produce a concentration of 50 (SD 3)%, as measured by the Capnomac sampling at the outflow of the anaesthetic machine.

A selection of eight isoflurane vaporizers from our operating theatre suite (Penlon Isotecs, Mk 3 or Mk 4) were checked for accuracy against the Capnomac at FGF of 1 and $6 \text{ l} \cdot \text{min}^{-1}$. Two minutes were allowed between changing the vaporizer setting and measuring the concentration in the FGF at the lower flow, but 30 s was found to be adequate for the concentration to stabilise at the higher flow rate. The temperature of the body of each vaporizer was measured using Ellab MHF probes (Ellab, Copenhagen) at the time of these tests. After all eight had been tested, the first vaporizer was tested again as a check for consistency. The time delay from moving the control knob on the vaporizer to measuring a change in the anaes-

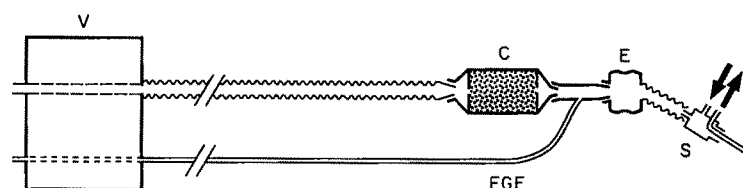


Fig. 1. The breathing system. A small canister charged with 160 g soda lime (C) connects with the patient's tracheal tube (not shown) via a port for the fresh gas (FGF), a catheter mount with integral heat and moisture exchanger (E), and a connector for gas sampling and return (S). Gas is drawn through a catheter from the tip of the tracheal tube. Mechanical ventilation is achieved by the ventilator (V) connected to the other end of the soda lime canister by a long corrugated tube of internal volume 2 l.

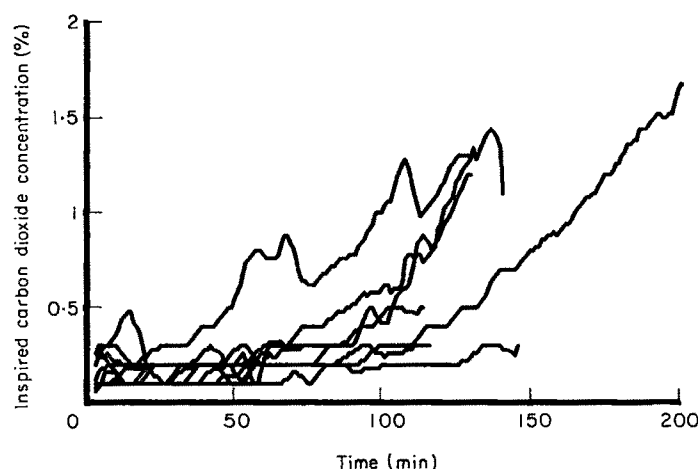


Fig. 2. Minimum inspired concentrations of carbon dioxide for 13 patients breathing via a small to-and-fro soda lime canister. Data have been sampled at 1 min intervals, and a running average taken over five consecutive data points in order to smooth the graphs.

thetic concentration in the FGF outlet of the anaesthetic machine was measured using a stop watch. Two anaesthetic machines were tested. In order to assess the time lag in the measurement technique (observer reflex time, sample transit time and Capnomac response time), the delays were also measured with a large FGF of $12 \text{ l} \cdot \text{min}^{-1}$. The analogue output of isoflurane concentration was recorded on the Lectromed Multitrace 4 pen-recorder to facilitate measurement of rise times.

We examined the possibility of contamination of the respiratory tract by soda lime dust. The fibre pad from the Edith HME becomes sufficiently wet in use to allow testing with universal pH paper, or the pH pad of a urine dip-stick (Multistix, Ames). The HME pads were extracted, cut into discs 1 cm thick, and tested for pH after four of the operations. The condensed liquid lying within the corrugations of the catheter mount was tested similarly. We also tested HME pads after the system had been used to ventilate a Manley Lung Performance Analyser (Blease). In this arrangement there is no carbon dioxide to neutralise soda lime before its presence can be tested. HME pads were extracted, cut into discs, wetted with water and tested with

universal pH paper after 5 min ventilation on two occasions, each time with new soda lime.

Results

The sex, age and weight of the patients is shown in Table 1 with some details of their anaesthetic.

Figure 2 illustrates the minimum inspired carbon dioxide concentrations after connection to the system. Only one procedure outlasted the absorptive capacity of the canister. In four cases the minimum inspiratory carbon dioxide concentration rose above 0.6%, but none of these required a minute volume greater than that used at the start of the procedure in order to maintain normocapnia.

The rise in inspired and end-tidal concentrations of nitrous oxide did not follow a simple exponential curve (Fig. 3). The mean (SD) times taken for inspired concentration to rise to 20% and 40% were 1.9 (0.8) min and 11.6 (5.2) min respectively (Table 1). Late in some of the procedures, the inspired concentration of nitrous oxide was noted to be slightly greater than that of the FGF. The

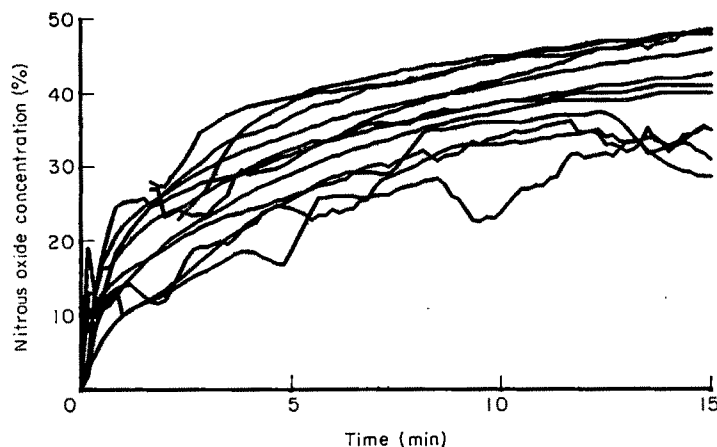


Fig. 3. The rise in nitrous oxide concentration in inspired gas using a to-and-fro breathing system, FGF $1 \text{ l} \cdot \text{min}^{-1}$ 50% nitrous oxide (case 12 excluded). Data were sampled at 10 s intervals and a running average taken over five consecutive data points in order to smooth the graphs.

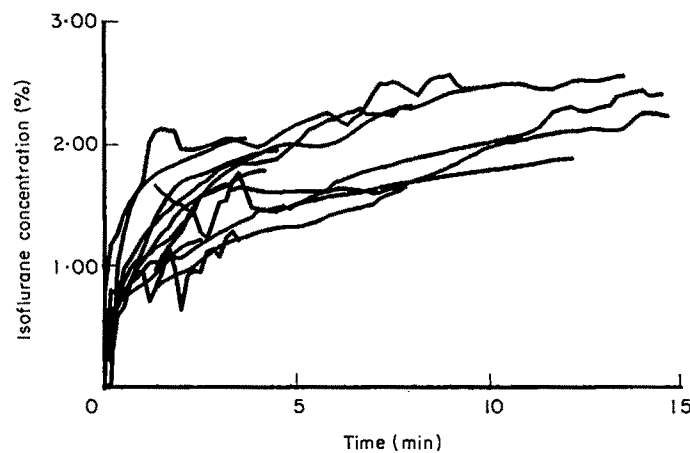


Fig. 4. The rise in isoflurane concentration in inspired gas using a to-and-fro breathing system, FGF $1 \text{ l} \cdot \text{min}^{-1}$ (case 12 excluded). Data was sampled at 10 s intervals and a running average taken over five consecutive data points in order to smooth the graphs.

greatest difference we observed occurred when the concentration in the FGF was 47% and the inspired concentration was 53%.

The time taken for the inspired concentration of isoflurane (C_I) to rise to 1% ranged from 1 to 3 min (mean 2.0 min). In eight cases C_{FGF} was maintained at 5% until C_I reached 2%, and this took from 4 to 14 min (mean 7.4 min). These results are illustrated in Figure 4 and Table 1. It was noticed that, for any particular patient, after the initial wash-in period the ratio of C_I to C_{FGF} was

remarkably constant. Instantaneous deviations from this ratio due to alterations of C_{FGF} lasted only a few minutes (Fig. 5). An asymptotic value of this ratio was estimated in each patient by simply laying a ruler over a graph of C_I/C_{FGF} against time, parallel to the time axis, and obtaining a best-fit by eye to a resolution of 0.5 (Fig. 6). The mean (SD) of these asymptotic ratios were 0.52 (0.07) at normocapnia. There was no correlation with patient age or weight (Table 1). Hyperventilation to an end-tidal carbon dioxide tension of 4 kPa produced a slight fall in the ratio, but never by more than 0.05. The alveolar concentration was unaffected by this degree of hyperventilation.

The wash out of isoflurane is shown in Figure 7. There is great variability between patients that cannot be explained solely by duration of anaesthesia.

The vaporizers were acceptably accurate for clinical work at both the FGF used in the tests (Fig. 8). All the vaporizers were in the temperature range 20.4–21.9°C. The difference between the initial and the repeated measurements on the first vaporizer were at most 0.1% absolute concentration (which is the resolution of the Capnomac display). The first readings only have been included in the

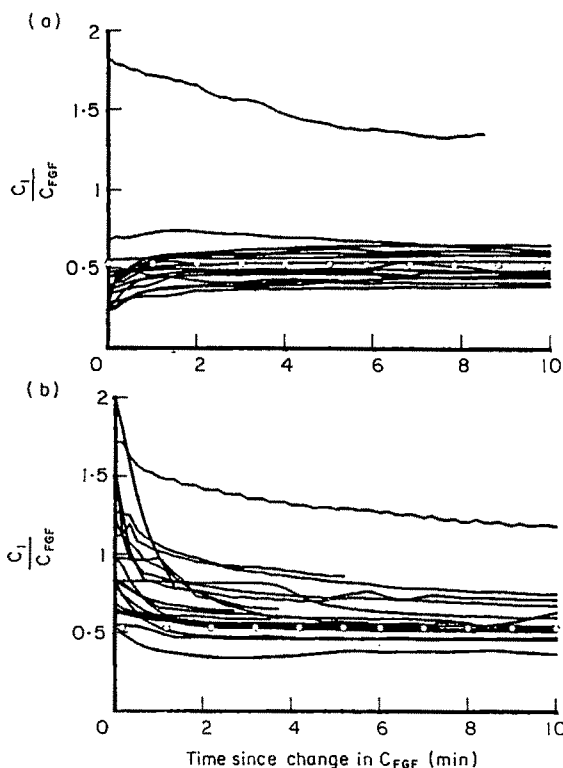


Fig. 5. The change in C_I/C_{FGF} following (a) an increase and (b) a decrease in C_{FGF} . Several curves may correspond to the same patient, and not all extend over 10 min because of intervening changes in C_{FGF} . The sizes of the changes in C_{FGF} , and the times at which they were made, are not significantly different between the two sets of data. The mean of the 'asymptotic ratios' (see Fig. 6 and text) is shown by the open circles.

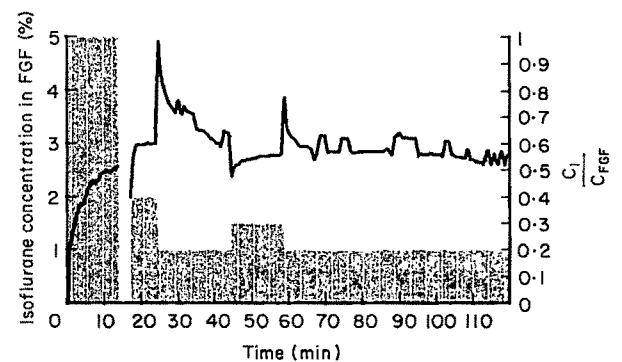


Fig. 6. The histogram shows the concentration of isoflurane in the fresh gas (left vertical axis) and the curve shows the value of the ratio C_I/C_{FGF} (right vertical axis). The asymptotic value of this ratio was determined by eye to a resolution of 0.05, excluding the period of wash-in, and periods of deliberate hyperventilation (75 to 86 min) and hypoventilation (86 to 91 min). In this instance the ratio was judged to be 0.6. (Data from case 10: the last 20 min of anaesthesia were conducted without isoflurane in the FGF).

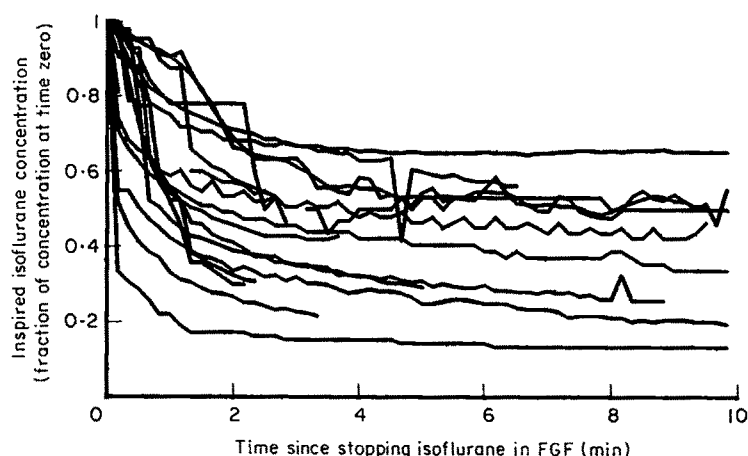


Fig. 7. The decline in inspired concentration of isoflurane after setting C_{FGF} to 0% while maintaining a FGF of 1 l.min^{-1} . Isoflurane concentrations are expressed as a fraction of the inspired concentration present at the time that isoflurane in the FGF was discontinued.

data presented here. The time lapse between turning the vaporizer knob and detecting a change in the analogue output of the Capnomac was 6 s with a FGF of 1 l.min^{-1} , and 2 s with a FGF of 12 l.min^{-1} (results rounded to the nearest second, with no variation between trials at this resolution). However, although the rise in C_{FGF} was virtually instantaneous when using the high FGF, the 0–90% rise time was 21 s with a FGF of 1 l.min^{-1} . The 100–10% fall time was 26 s. When the vaporizer knob was turned from off to 1%, an excess of isoflurane was commonly delivered for a few seconds, particularly if the vaporizer had not been in use for a little while. This effect was less obvious at the high FGF (commonly producing a momentary concentration of 2%) than at the lower FGF (commonly reaching 4% with a small excess still detectable 1 min later). The quantity of excess delivered as a bolus was greater at the lower FGF: the largest bolus was observed was about 5 ml of isoflurane vapour, of which 3 ml was delivered in the first 10 s.

The pH of fluid found in the corrugations of the Edith catheter mount was consistently found to be 6. The results of the examination of all HME pads (those used on patients and those used with the Lung Performance Analyser) were identical: an alkaline reaction was found only on testing the surface of the HME element facing the soda lime canister. All samples taken from the interior of the element showed a pH of 6 or 7 on the dipsticks and universal pH paper.

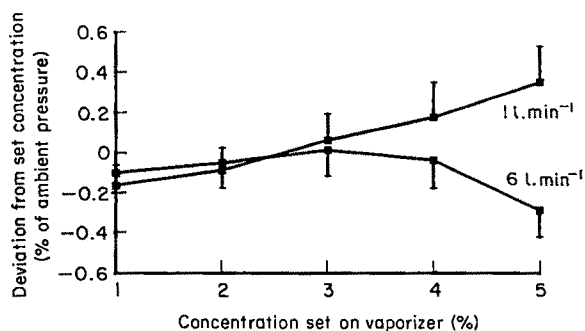


Fig. 8. The mean (2 SEM) accuracy of an arbitrary selection of eight Penlon Isotec Mk 3 and 4 vaporizers at two fresh gas flows.

Discussion

We have detected a distrust of low-flow anaesthesia among the new generation of anaesthetists, trained to use high-flow systems which provide a constant inspired concentration of anaesthetic agent. Few have any experience of low-flow anaesthesia, and those that have, have generally used the circle system. The advantages of reduced cost, reduced pollution, and improved heat and water conservation, are generally acknowledged, but are not considered sufficient to change practice. The arrival of a very expensive new agent may change the balance of judgement.

We believe that little experience is required to become confident with the system described. Very little new equipment is needed. Standard vaporizers are good enough to use at this fresh gas flow (i.e. 1 l.min^{-1}), and the excess concentration produced when first turned on will produce a negligible rise in the inspired concentration. We have not found our small canister to be at all inconvenient. Our laboratory investigation [1] and this trial lead us to expect the soda lime to last for 2 h in the majority of patients whose lungs are being ventilated. A low concentration of carbon dioxide in inspired gas is of no consequence when the lungs are ventilated mechanically because the alveolar ventilation can be increased easily if necessary, although it would be an undesirable burden in a patient ventilating spontaneously. We would not want the minimum inspired carbon dioxide concentration in such a patient to rise as we have seen in this study. The colour indicator was helpful to determine exhaustion of soda lime, but ideally continuous capnography would be used. This is now available in many hospitals. The soda lime canister can quickly be changed; this has no adverse effect on the course of the anaesthetic, although alternatively the FGF could be increased to diminish or prevent rebreathing. The HME element in the Edith catheter mount is an effective barrier to soda lime dust, and although the size of canister does not seem to generate high temperatures, the HME will also protect the patient from excessively high temperatures should they occur [1].

We anticipate a period of at least 10 min before inspired oxygen and nitrous oxide concentrations could be assumed to be close to those in the fresh gas, even if anaesthesia is induced using a high flow system. During this time nitrous oxide uptake is rapid [5] and it concentrates the oxygen in

the breathing system, but later — when the rate of uptake of nitrous oxide falls below that of oxygen — the reverse effect is seen. Our results show that the nitrous oxide concentration rises more rapidly than has been predicted by computer simulation [6]. Even though we have not seen excessive concentrations of nitrous oxide in the inspired gas, we would not be happy to use less than 35% oxygen in the FGF.

A period of 5 or 10 min is also required before the inspired concentration of isoflurane can be predicted easily. We have found consistently that, after this initial period of wash-in, the inspired concentration of isoflurane becomes approximately half that in the FGF within a few minutes of any adjustment (Fig. 5), so although an anaesthetic analyser is desirable, it is not essential. (We think that the error introduced by this rule of thumb is small compared to, say, the error in the average anaesthetist's estimation of alveolar isoflurane concentration when using a high-flow system which delivers a known inspired concentration.) When C_{FGF} is increased, C_i/C_{FGF} is instantaneously reduced and in general is less than 0.5. Converse changes occur when C_{FGF} is decreased. Exceptions occur: if increases and decreases in C_{FGF} are made in rapid succession then hybrid effects are seen in C_i/C_{FGF} , and if the reduction in C_{FGF} is large and made after a long period of anaesthesia then C_i/C_{FGF} declines more slowly to its previous value because of the coasting effect. In general, changes in C_i/C_{FGF} caused by a reduction in C_{FGF} take longer to settle than changes due to an increase (Fig. 5). On one occasion the finding of a low ratio (less than 0.3) prompted the discovery of an error in the FGF settings — only 700 ml.min⁻¹ was being delivered. These data are excluded from those presented here, but the case illustrates the reliability of the rule that C_i is approximately half of C_{FGF} .

If the inspired mixture is assumed to be a perfect mixture of fresh, dead space and alveolar gas we can obtain a simple theoretical estimate of C_i from the following equation:

$$\dot{V}_{FGF} \cdot C_{FGF} + \dot{V}_A \cdot C_A = (\dot{V}_{FGF} + \dot{V}_A) \cdot C_i$$

(Expired dead space gas has the same composition as inspired gas, and a term $(\dot{V} - \dot{V}_A) C_i$ cancels from each side of the equation.) Assuming an alveolar ventilation of 4 l.min⁻¹, and using the fact that the alveolar concentration of isoflurane (C_A) is approximately 0.7 C_i after the first half hour of anaesthesia [7], then $C_i/C_{FGF} = 0.45$. This is close to the mean of the asymptotic ratios found in our patients. The simple model correctly predicts the qualitative, although not the quantitative, effect of ventilatory changes. We are therefore not entirely surprised to find the ratio C_i/C_{FGF} so constant in time.

During the wash-out period (Fig. 7), typical coasting is seen until the FGF is increased: the inspired concentration falls rapidly in the first few breaths and more slowly thereafter. However, we have been unable to correlate the patient's age, weight or duration of anaesthesia with either the magnitude of the initial fall, or the rate of the subsequent decline.

The delay in response of C_i to changes in C_{FGF} is unquestionably a disadvantage in comparison with high-flow systems, and care is required to ensure that any inherent and inevitable delay between vaporizer setting and change in C_{FGF} (at the anaesthetic machine) is not compounded by using a supply pipe for the FGF of large internal volume. It

might be expected that a to-and-fro system would respond more quickly to changes in the composition of the FGF than a circle system, which has a large internal volume to act as buffer. We have monitored and recorded routine anaesthetics using a circle system (BOC Mk IV) with a FGF of 1 l.min⁻¹, but have not observed any functional differences of clinical significance between the two systems apart, of course, from the longer duration of carbon dioxide absorption with the circle system. For instance, the mean (SD) of the asymptotic ratio of C_i to C_{FGF} in seven patients was 0.52 (0.09), exactly the same value we found with the to-and-fro system. (We believe that the value of this ratio depends mainly on the magnitude of the FGF and the choice of anaesthetic agent, not on the design of the breathing system.) In fact, the only consistent differences we have found has been in the relationship between C_i and C_A after the isoflurane in the FGF has been discontinued. In both systems the two concentrations start reducing immediately and become equal within a few minutes. However, with the to-and-fro system C_i falls below C_A within a few breaths and remains less than C_A until they become equal; with the circle system, C_i remains greater than or equal to C_A at all times. We believe this difference to be of academic interest only, for C_A reduces at the same rate in both systems.

The major reward to come from the use of a low flow system is financial. The current cost of anaesthetic agents in a FGF delivering 50% nitrous oxide and 1% isoflurane at 6 l.min⁻¹ is £6.69 per hour. The cost of anaesthetic required to produce a similar inspired concentration in a low-flow system (i.e. a FGF of 50% nitrous oxide and 2% isoflurane at 1 l.min⁻¹) is £2.10, to which must be added the £0.35 cost of soda lime. The soda lime costs the equivalent of 3 min anaesthesia using the high-flow system, so savings can be made on anaesthesia for even the shortest procedures. We have used Engstrom Edith HME catheter mounts because they are routinely available in our hospital. Although their use is desirable in a high-flow system, a dust trap is essential in a to-and-fro system and it may be considered as a minimum of £1 extra to the cost of every low-flow anaesthetic. However, even allowing for the continued use of high-flow systems in the anaesthetic room, there could still be a 50% saving in the use of nitrous oxide and isoflurane. We expect such a saving to be particularly important when desflurane becomes available.

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Changes in arterial blood pressure during traction of extra-ocular muscles. A study in rabbits

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Summary

A significant reflex fall in mean arterial blood pressure from baseline values was observed during extra-ocular muscle traction in rabbits. This effect could be abolished only by retrobulbar block (afferent pathway block) and not by vagotomy, intravenous atropine or glycopyrronium, suggesting that it is distinct from, and independent of, the oculocardiac reflex. We suggest that these reflex changes in blood pressure be known as the 'oculodepressor reflex'.

Key words

Anaesthesia; ophthalmic.

Autonomic reflexes; oculocardiac, oculorespiratory.

Reflex changes in heart rate and/or cardiac rhythm during extra-ocular muscle traction are well documented in the literature and constitute the 'oculocardiac reflex' (OCR) [1]. Similarly, reflex changes in respiratory rate and/or rhythm have been designated the 'oculorespiratory reflex' (ORR) [2].* Reflex changes in arterial blood pressure have not been extensively studied to date and this aspect forms the basis of the present experimental work. The relationship between alterations in blood pressure and the oculocardiac reflex has also been studied.

Methods

The experiments were carried out on 20 albino rabbits of either sex of 1–2 kg body weight. After fasting overnight the rabbits were anaesthetised with intravenous pentobarbitone (40 mg.kg⁻¹). Tracheostomy was performed and the vagi were exposed in the cervical region. A femoral vein was cannulated for intermittent injections and the infusion of anaesthetic drugs. The femoral artery on the same side was also cannulated and connected to a pressure transducer which in turn was connected to one channel of a four channel recorder (Polyrite, INCO). Respiratory values were recorded from the tracheal cannula by a separate trans-

ducer on the second channel. The electrocardiogram (ECG) was recorded by needle electrodes.

The rectus muscles, medial (MR), lateral (LR), superior (SR) and inferior (IR), were exposed by a gently performed perilimbal peritomy and a silk suture loop was passed under each muscle. A silk suture with wire hooks on both ends was passed over a pulley fixed on a stand. One hook was engaged in the silk suture loop under the muscle tendon and the other was used to attach weights for the mechanical stimulus.

In each rabbit, after taking basal recordings of arterial blood pressure and ECG, traction with 150 g was applied by square wave (SW) stimulus (acute traction sustained for a minimal period of 20 s followed by acute release) to the medial rectus, and changes in arterial blood pressure, heart rate and rhythm were recorded. The procedure was repeated for the lateral, superior and inferior recti after a pause of 3 min in each case.

For further study the 20 rabbits were divided into four equal groups. In group 1, after taking the basal recordings, bilateral vagotomy was performed in the cervical region and its effects on blood pressure and OCR were elicited by the square wave stimulus to the medial rectus only. Similarly, the modifying effects of intravenous atropine (15 µg.kg⁻¹) glycopyrronium (7.5 µg.kg⁻¹) and retrobulbar block (10 min after injection of 2 ml 2% lignocaine) were examined in rabbits of groups 2, 3 and 4, respectively.

A decrease in heart rate by more than 20% from basal

*The clinical manifestations of the ORR include bradypnoea, gasping, inspiratory pause, shallow breathing and respiratory arrest.

Table 1. Decrease (SD) in mean arterial blood pressure (MAP) during extra-ocular muscle traction.

Animals	n	MAP (mmHg)			
		Basal	Peak muscle traction	Absolute decrease	Percentage decrease
All	20	89 ^x (9.9)	49 ^y (14.4)	40 (18.0)	45 ^a (17.4)
Positive OCR	14	86 (9.1)	47 (15.5)	39 (18.6)	45 ^b (18.6)
Negative OCR	6	97 (7.2)	52 (10.9)	45 (16.0)	46 ^c (14.2)

p value, x versus y = < 0.001; a versus b = > 0.10; b versus c = > 0.10 and a versus c = > 0.10.

values and/or occurrence of arrhythmia during traction on an extra-ocular muscle constituted a positive OCR [3].

Results

The mean (SD) basal heart rate and mean (SD) arterial blood pressure (MAP) observed were 273 (44) beat.min⁻¹ and 89 (10) mmHg, respectively. The MAP decreased significantly from baseline values (p < 0.001) within 2 s of the application of the SW stimulus in all the animals studied while a positive OCR was observed in 14 (70%) animals (Table 1). The mean percentage falls in MAP observed in animals with positive or negative OCRs were almost identical (Table 1). Similarly, the degree of hypotension observed during traction of different recti was very similar (Table 2). A positive OCR was observed in 14 (70%) animals during MR stimulation compared with seven (35%) each with LR, SR and IR muscles.

The intravenous administration of atropine or glycopyrronium or bilateral vagotomy before muscle traction, decreased the incidence of OCR by 100, 100 and 75%, respectively (Table 3). None of these altered the significant fall in arterial blood pressure. In contrast, retrobulbar block prevented both the hypotension and the OCR in all the animals studied (Table 3). The effects of vagotomy, atropine, glycopyrronium and retrobulbar block on altera-

Table 3. Frequency of oculocardiac (OCR) and oculodepressor reflex (ODR) during medial rectus traction after the treatments shown.

Treatment	n	Positive OCR		Positive ODR	
		Before	After	Before	After
Bilateral vagotomy (Group 1)	5	4	1	5	5
Atropine; 15 µg.kg ⁻¹ (Group 2)	5	3	0	5	5
Glycopyrronium; 7.5 µg.kg ⁻¹ (Group 3)	5	4	0	5	5
Retrobulbar block; (Group 4)	5	3	0	5	0

tions in mean arterial pressure during extraocular muscle traction are shown in Table 4.

The graphical recording of reflex hypotension obtained during medial rectus traction is shown in Figure 1. Figures 2 to 5, depict effects of vagotomy, atropine, glycopyrronium and retrobulbar block on the OCR and reflex hypotension, respectively.

Discussion

The oculocardiac reflex was first described by Aschner in 1908 [4] and has been extensively studied since. The significant fall in blood pressure observed in the present experiments during extra-ocular muscle traction has not been reported in the available literature. One might expect the cardiac output and therefore arterial blood pressure to fall in the presence of significant cardiac slowing due to OCR. However, the identical hypotension observed in animals with positive or negative OCRs suggests that the alterations in arterial blood pressure are independent of rate. Furthermore, significant hypotension in the absence of bradycardia i.e. after abolition of OCR by vagotomy, atropine and glycopyrronium (Figs. 2, 3 and 4) strongly indicates that the fall in blood pressure observed on extra-ocular muscle stimulation may not be related to OCR. Since passive stretching of the extra-ocular muscles

Table 2. Mean (SD) decrease in mean arterial blood pressure (MAP) during traction on different extra-ocular muscles.

Muscle stretched	n		MAP (mmHg)			
	All	With positive OCR	Basal	During peak effect of muscle traction	Decrease	Percentage decrease
Medial rectus	20	14	89 (9.9)	49 (14.4)	40 (18.0)	45 ^a (17.4)
Lateral rectus	20	7	92 (9.4)	53 (14.5)	39 (17.0)	42 ^b (17.7)
Superior rectus	20	7	93 (10.5)	56 (14.7)	37 (21.3)	40 ^c (19.5)
Inferior rectus	20	7	89 (12.6)	52 (14.3)	37 (20.5)	42 ^d (19.6)

p value, a versus b = > 0.10; a versus c = > 0.10; a versus d = > 0.10; b versus c = > 0.10; b versus d = > 0.10 and c versus d = > 0.10.

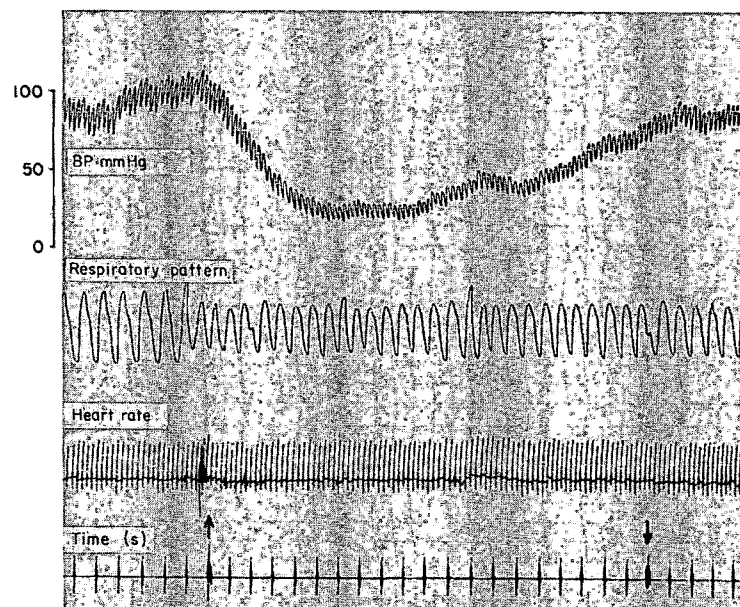


Fig. 1. Changes in heart rate, respiratory pattern and arterial blood pressure during extra-ocular muscle traction. $\uparrow$ = start of stimulus; $\downarrow$ = end of stimulus.

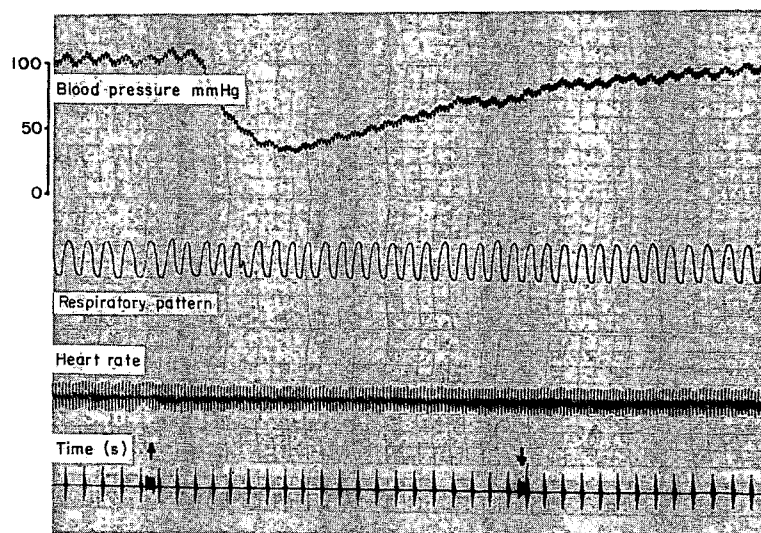


Fig. 2. Effect of bilateral vagotomy on heart rate, respiratory pattern and arterial blood pressure during extra-ocular muscle traction. $\uparrow$ = start of stimulus; $\downarrow$ = end of stimulus.

Table 4. Mean (SD) decrease in mean arterial blood pressure during medial rectus traction after the treatments shown.

		MAP (mmHg)							
		Before				After			
Treatment	n	Basal	During peak effect of muscle traction	Decrease	Percentage decrease	Basal	During peak effect of muscle traction	Decrease	Percentage decrease
Vagotomy (Group 1)	5	78 ^a (7.4)	50 ^b (12.1)	28 (7.2)	37 (11.5)	85 ^{a'} (9.8)	58 ^{b'} (9.5)	27 (14.1)	31 (12.7)
Atropine; 15 $\mu\text{g.kg}^{-1}$ (Group 2)	5	97 ^c (4.0)	43 ^d (17.9)	54 (16.1)	56 (17.7)	98 ^{c'} (8.3)	54 ^{d'} (12.1)	44 (15.4)	44 (14.0)
Glycopyrronium; 7.5 $\mu\text{g.kg}^{-1}$ (Group 3)	5	87 ^e (15.4)	51 ^f (19.3)	36 (19.3)	40 (19.5)	87 ^{e'} (8.0)	51 ^{f'} (11.2)	36 (14.2)	41 (14.8)
Retrobulbar block; (Group 4)	5	95 ^g (5.6)	50 ^h (9.1)	45 (16.5)	47 (13.8)	89 ^{g'} (8.2)	87 ^{h'} (7.4)	2 (1.7)	1 (1.9)

p value, a versus b = < 0.001; a' versus b' = < 0.001; c versus d = < 0.001; c' versus d' = < 0.001; e versus f = < 0.001; e' versus f' = < 0.001; g versus h = < 0.001 and g' versus h' = > 0.10.

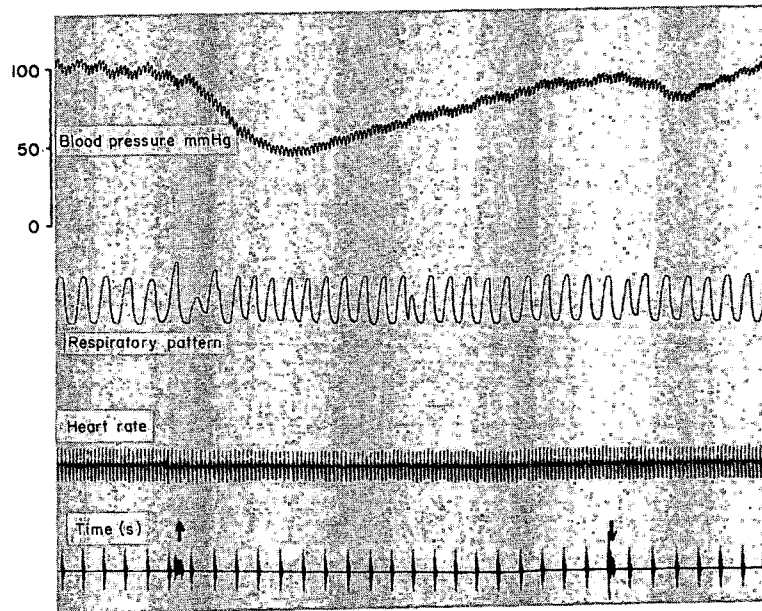


Fig. 3. Effect of atropine on heart rate, respiratory pattern and arterial blood pressure during extra-ocular muscle traction. $\uparrow$ = start of stimulus; $\downarrow$ = end of stimulus.

produced hypotension immediately within 2 s which disappeared on release, this response appears to be reflex in nature. This reflex hypotension seems to be distinct from, and independent of, the OCR, and we suggest that it be called 'the oculodepressor reflex' (ODR).

There were no significant differences in the hypotension observed on stimulation of different extra-ocular muscles ($p > 0.10$) (Table 2). However, the incidence of OCR observed with stimulation of medial rectus was higher than that with the lateral, superior and inferior rectus muscles, and supports the findings of other workers [5, 6].

The occurrence of OCR and fall in blood pressure (ODR) on extra-ocular muscle traction could be prevented in 100% of the animals by retrobulbar block (Tables 3 and 4; Fig. 5). This observation supports the views of other workers who have suggested that the retrobulbar injection

of local anaesthetics blocks the afferent pathway of the OCR [5, 6]. This finding also suggests that the afferent pathways of the OCR and ODR may be similar. However, the abolition of OCR with vagotomy, atropine or glycopyrronium suggests that the efferent pathways of the two reflexes must be different.

The afferent pathway of the OCR was described by Aschner [4]. It consists of fibres which run with short ciliary nerves to the ciliary ganglion and then with the ophthalmic branch of the trigeminal nerve to the Gasserian ganglion. From there the fibres reach the main sensory nucleus of the trigeminal nerve and are relayed to the vagal nucleus by short internuncial fibres in the reticular formation. Gay and colleagues [7] concluded that the afferents mediating the ORR in the cat pass with IIIrd, IVth and VIth cranial motor nerves intra-orbitally, leaving these to join the

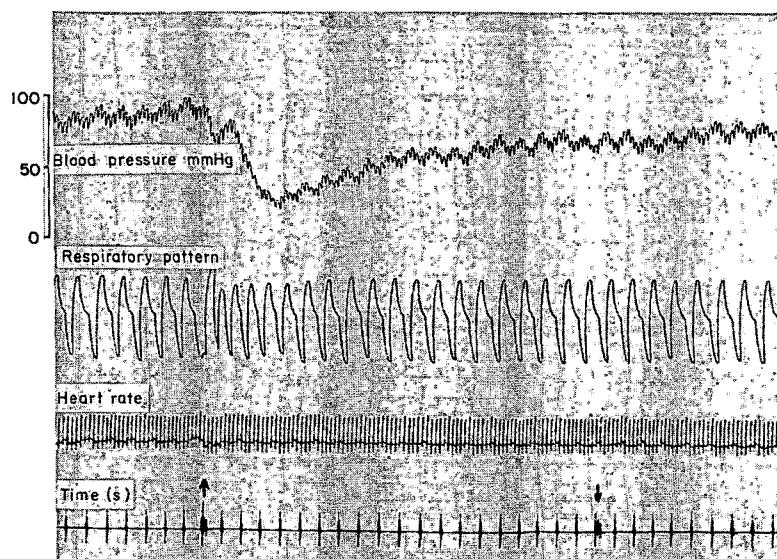


Fig. 4. Effect of glycopyrronium on heart rate, respiratory pattern and arterial blood pressure during extra-ocular muscle traction. $\uparrow$ = start of stimulus; $\downarrow$ = end of stimulus.

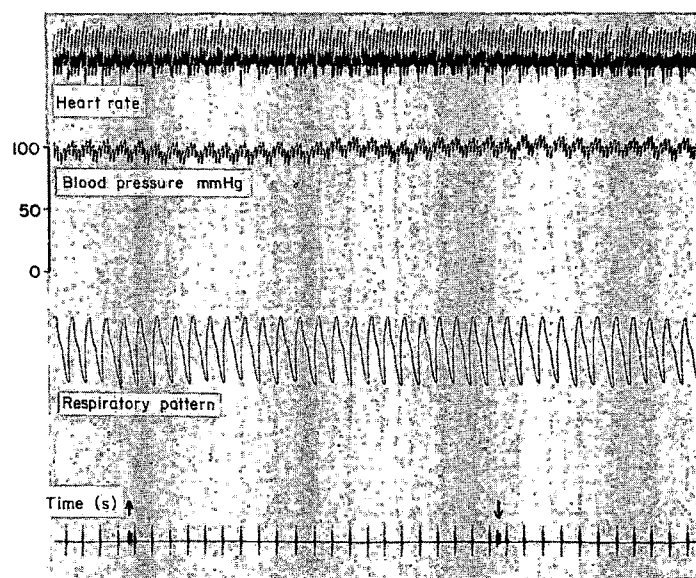


Fig. 5. Effect of retrobulbar block on heart rate, respiratory pattern and arterial blood pressure during extra-ocular muscle traction. ↑ = start of stimulus; ↓ = end of stimulus.

ophthalmic division of the trigeminal nerve before it leaves the cavernous sinus. The initial afferent pathway of the ODR is not known but in the light of the above it seems likely that the stimulus is relayed through the sensory nucleus of the trigeminal nerve possibly to the vasomotor centre leading to reflex hypotension by inhibition of efferent vasosympathetic nerves. However, this hypothesis clearly requires experimental verification.

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CASE REPORT

Hyperkalaemic periodic paralysis and anaesthesia

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Summary

Hyperkalaemic periodic paralysis is the rarer of the two forms of potassium-associated familial paralysis. We report a family with hyperkalaemic periodic paralysis with paramyotonia and the anaesthetic management of four affected members. In three of these, paralytic episodes had been precipitated by previous anaesthesia, but this was avoided in the anaesthetics described. We conclude from our experiences that with depletion of potassium before surgery, prevention of carbohydrate depletion, avoidance of potassium-releasing anaesthetic drugs and maintenance of normothermia, patients with hyperkalaemic periodic paralysis can be anaesthetised without complications. We have no evidence that they exhibit abnormal sensitivity to nondepolarising neuromuscular relaxants.

Key words

Complications; hyperkalaemic periodic paralysis, paramyotonia.

The periodic paralyses are a group of familial myopathic disorders characterised by episodic flaccid weakness associated with changes in serum potassium. The first case report appeared in 1882 [1] but it was only in this century that the association with potassium changes was recognised [2]. The early case reports were of episodic weakness associated with hypokalaemia. In 1956, Gamstorp *et al.* [3, 4] described a condition that was clinically very similar, but characterised by elevated serum potassium concentrations during the attack. She described 138 affected individuals in two families and named the disorder *adynamia episodica hereditaria* (now known as hyperkalaemic periodic paralysis or HPP) [4], but she did not describe any myotonic symptoms in these patients. The hyperkalaemic form of periodic paralysis is much rarer than the hypokalaemic form, and three clinical variants of hyperkalaemic periodic paralysis are now recognised: HPP with myotonia, HPP without myotonia and HPP with paramyotonia (cold-induced myotonia). The family described here showed the third variant.

Paramyotonia congenita was described by Eulenberg in 1886 [5]. It has autosomal dominant inheritance and is characterised by myotonia precipitated by exposure to cold and made worse by exercise. This is different from the myotonia of myotonic dystrophy. 'Pure' paramyotonia has been reported in some families in West Germany, but many

other families which exhibit a combination of symptoms of episodic weakness and paramyotonia have been reported [6–12]. A link between hyperkalaemic periodic paralysis and paramyotonia congenita was first suggested by Drager *et al.* [6], since both conditions show hyperkalaemia, and this association has been the subject of continuing debate. The fact that there are families in whom symptoms of both disorders coexist and that both disorders have similar abnormalities of sodium channel function, lends support to the hypothesis that they are variants of the same disease.

Patch clamp techniques have been used to investigate muscle fibres of patients with HPP and various other myotonic disorders, and it was previously thought that the myotonia resulted from decreased calcium conductance secondary to a defect within the sarcolemma. This has been demonstrated in myotonic goats and humans with myotonia congenita. However, in patients with paramyotonia congenita and *adynamia episodica* with myotonia, the sodium currents are affected [13].

During an attack of weakness, the muscle fibres of a patient with HPP are depolarised to such an extent that the membrane is inexcitable. This membrane depolarisation is caused by a pathological inward sodium current, conducted by noninactivating sodium channels [14, 15].

Genetic linkage studies in several large families in

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Germany, America and the UK who have HPP with myotonia, indicate that the sodium channel alpha-subunit gene on chromosome 17 contains the HPP mutation [14, 15]. Subsequently, families with paramyotonia congenita have shown linkage to the same gene locus, which indicates that hyperkalaemic periodic paralysis and paramyotonia congenita must be considered as one disease entity (personal communication, K. Ricker).

The anaesthetic literature on these conditions, particularly the rarer hyperkalaemic form, is sparse. Egan and Klein in 1959 described three families with HPP [16]. At least one member of each family had received general anaesthesia and in each case, on awakening, the patient had a paralytic episode lasting several hours. Another member of one family later received an uneventful spinal anaesthetic for child birth.

In 1980, Flewellen and Bodenstener described a case of successful anaesthesia in a 10-year-old girl with HPP [17]. Katz *et al.* summarised the limited knowledge and recommended avoidance of carbohydrate depletion by dextrose infusion and ECG monitoring for signs of hyperkalaemia. They considered the use of neuromuscular relaxants to be contraindicated because of possible sensitivity [18] although there is no evidence to support this. Aarons *et al.* used a nondepolarising neuromuscular relaxants in an adult patient with HPP who experienced no postoperative weakness [19].

We report a family with hyperkalaemic periodic paralysis with paramyotonia. Four members of this family underwent successful general anaesthesia, which included the use of neuromuscular relaxants, for abdominal surgery. Three out of the four patients had previously had problems with anaesthesia which were related to their disease.

The disease, which has autosomal dominant inheritance with almost complete penetrance, has been reported in five generations of this particular family. It is thought to have been introduced to Australia from Sweden, by the maternal grandfather of the third and fourth patients described below.

Case histories

This family's condition was definitively diagnosed in 1980 when the proband, a 38-year-old male, presented to a neurologist after problems at induction of anaesthesia for an elective cholecystectomy. Anaesthesia was aborted because of an episode of marked generalised muscle stiffness and a locked jaw which occurred following thiopentone and suxamethonium. For several days afterwards, he had profound global weakness, but without respiratory involvement, and muscle pains. Similar, but infrequent episodes of global paralysis had occurred between the ages of 5 and 12 years. In addition, the patient described difficulty in relaxing his muscles on initiating movements, and muscle stiffness which was more marked in cold weather. He then remained symptom free until the episode precipitated by this anaesthetic.

His mother and several other family members were similarly affected (Fig. 1). Full investigation, which led to the diagnosis of HPP with paramyotonia, was carried out in 1980. Electromyographic (EMG) studies revealed widespread myotonic discharges, which were marked in the lower limbs and greatly augmented by cold. A provocation test with an oral potassium load precipitated an episode of

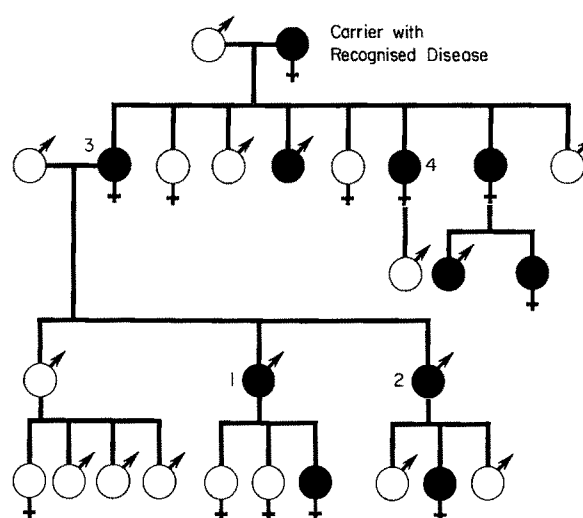


Fig. 1. Family tree of four generations showing 11 affected individuals in a total of 27 persons at risk. The numbers assigned to the four patients reported in this article are according to the order of presentation to the Anaesthesia Department. Those subjects known to be affected are shown as filled circles.

generalised weakness which was reversed with intravenous calcium gluconate. The value of calcium in reversing weakness in HPP has been reported by van't Hoff [6] and Gamstorp [10]. A muscle biopsy was performed, which revealed nonspecific myopathic changes, and biochemical studies showed an increased serum creatine kinase concentration (CK) and unconjugated hyperbilirubinaemia. Elevated CK has been previously described in the periodic paralyses. The hyperbilirubinaemia in this patient was due to the incidental coexistence of Gilbert's syndrome, although it had in fact been the indication for the proposed cholecystectomy.

Case one

Two years after the diagnosis had been made, the younger brother of the proband, aged 37 years, presented with biliary colic and was scheduled for cholecystectomy. He gave a similar history to that of his brother. In addition, 19 years previously, anaesthetic complications had occurred during an inguinal hernia repair. He had developed muscle 'spasm' after suxamethonium and repeated doses had produced no relaxation. There were no further anaesthetic details, but marked muscle weakness had occurred for 10 days after surgery.

The patient's general health was good and pre-operative ECG and routine blood tests were normal. Serum potassium concentration was 4.1 mmol.l^{-1} before surgery. He was receiving procainamide, which he took before exercise, to prevent myotonia. Procainamide, a type I anti-arrhythmic agent that affects sodium influx during depolarisation, has been shown to be useful in the treatment of myotonia. Other type I anti-arrhythmic drugs have similarly been found useful [18, 19].

After consultation with the neurologist, the peri-operative management was planned. Frusemide 40 mg daily was given for 3 days before surgery. Premedication was with lorazepam 3.75 mg 4 h pre-operatively. Induction was with thiopentone and incremental doses of pancuronium were given, monitored with a nerve stimulator until adequate

myoneural blockade was achieved. A total dose of 0.1 mg.kg^{-1} was given. The trachea was then intubated and anaesthesia maintained with nitrous oxide, oxygen and enflurane, supplemented with morphine 15 mg intravenously. Droperidol 3 mg was given for its antiemetic effect. Monitoring was with an ECG, pulse monitor, noninvasive blood pressure, spirometer and a peripheral nerve stimulator. A warming blanket was used and 1000 ml of warmed dextrose 5% was given. No further pancuronium was required and after 80 min , neuromuscular blockade was reversed with atropine 1.8 mg and neostigmine 5 mg . Emergence was unremarkable, the tracheal tube was removed and the patient had no postoperative weakness or muscle pain. A muscle biopsy showed nonspecific changes of a primary myopathy.

Case two

Three years after this, the propositus himself was rescheduled for cholecystectomy. No attacks of weakness had occurred since the previous anaesthetic and he was taking calcium supplements as a preventive measure. Pre-operative haematological and biochemical investigations and ECG were normal and the serum potassium concentration was 3.8 mmol.l^{-1} . Two doses of frusemide were given to deplete the body potassium stores, one the evening before and the other the morning of surgery. Premedication was with papaveretum 20 mg and metoclopramide 10 mg intramuscularly 1 h before surgery. Induction was with thiopentone and the trachea was intubated after atracurium 0.5 mg.kg^{-1} . Anaesthesia was maintained with 70% nitrous oxide in oxygen and enflurane and supplemented with morphine 4 mg . A warmed, balanced salt (compound sodium lactate) solution (500 ml) was given intra-operatively followed by dextrose in saline after surgery. Throughout anaesthesia the ECG, blood pressure, oesophageal temperature, oxygen saturation and end-tidal carbon dioxide concentration were monitored and a peripheral nerve stimulator was used. After 50 min , myoneural blockade was reversed with atropine 1.2 mg and neostigmine 2.5 mg . Spontaneous respiration returned and the tracheal tube was removed. No postoperative weakness, was experienced by the patient.

Case three

The mother of the first two patients had developed myotonic symptoms at the age of 17 years and these would be precipitated by involvement of a limb in an unusually intense or novel activity. Her myotonic symptoms were much more intense in the cold. In addition, the patient had experienced several episodes of global weakness. After the delivery of each of her three children and following surgery in 1962 (cholecystectomy and hernia repair) she had profound weakness lasting many days but not involving respiratory muscles. Several minor surgical procedures had been followed by a general weakness, despite the fact that the use of myoneural blockers had been avoided. In addition, during a gaseous induction with halothane for one of these, a significant bradycardia, which responded to atropine, had occurred. A similar event had taken place after induction with fentanyl and midazolam, during which the

pulse rate decreased to 28 beat.min^{-1} . However, on both occasions the patient was receiving atenolol and chlorothiazide for hypertension and timolol eye drops for glaucoma.

She had gastro-oesophageal reflux and required anti-reflux surgery in 1990 . Haematological and biochemical investigations were normal (serum potassium 3.6 mmol.l^{-1}) and her ECG showed sinus bradycardia (50 beat.min^{-1}) with some junctional escape beats.

In view of her chronic thiazide therapy, it was felt that no additional potassium depletion was indicated. An infusion of dextrose 5% was started after breakfast. Premedication was with temazepam 10 mg , metoclopramide 10 mg and ranitidine 150 mg orally, 2 h before afternoon surgery.

On arrival in the operating room, ECG, noninvasive blood pressure and pulse oximetry were established. The patient's lungs were pre-oxygenated and anaesthesia induced with thiopentone and vecuronium 0.1 mg.kg^{-1} . Cricoid pressure was applied as consciousness was lost and intermittent positive pressure ventilation (IPPV) by mask with nitrous oxide in oxygen and enflurane was commenced. The trachea was intubated and anaesthesia was supplemented with 10 mg of morphine. In addition, the end-tidal CO_2 concentration, oesophageal temperature and degree of neuromuscular blockade were monitored.

Twenty minutes after induction the patient developed a bradycardia of 30 beat.min^{-1} which responded to atropine. The serum potassium concentration was measured, although the ECG did not suggest hyperkalaemia, and was found to be 3.3 mmol.l^{-1} . After 60 min , the first twitch response reappeared and 90 min after induction the myoneural blockade was readily reversed with atropine 1.2 mg and neostigmine 2.5 mg . Spontaneous ventilation returned and the trachea was extubated without problem.

During surgery, 1000 ml dextrose 5% and 500 ml saline 0.9% were given via a blood warmer. In addition, a water warming blanket and a heat and moisture exchanger in the breathing circuit were used. The legs and head were wrapped in plastic to help prevent heat loss.

Postoperatively the patient had no muscle pain and despite a very minor degree of weakness in her back muscles she was able to sit and mobilise without difficulty.

Case four

The maternal aunt of the propositus first presented for surgery, an elective cholecystectomy, in 1991 . She was aware of the family history, but was less severely affected than other members and was receiving no therapy despite episodes of muscles stiffness and weakness.

Pre-operative investigations were normal, with a serum potassium concentration of 3.9 mmol.l^{-1} . Frusemide, 40 mg on the night before and a further 40 mg on the morning of surgery, was given for its kaliuretic effect and dextrose 5% infusion was started once fasting had begun. Premedication was with temazepam 20 mg and metoclopramide 10 mg .

The patient arrived in the operating room relaxed and drowsy. Noninvasive blood pressure measurement, ECG and pulse oximetry monitoring were established. The patient's lungs were pre-oxygenated and anaesthesia induced with thiopentone and atracurium 0.5 mg.kg^{-1} . The trachea was intubated and anaesthesia maintained with

70% nitrous oxide in oxygen and enflurane and supplemented with 10 mg of morphine intravenously.

Fasciculations of the abdominal muscles were noted during incision but the ECG did not suggest a rise in serum potassium concentration and a normal level was confirmed by laboratory testing. The muscles appeared adequately relaxed, despite these fasciculations.

Thirty minutes after induction the patient had two out of four twitches in response to train-of-four nerve stimulation and a further 10 mg bolus of atracurium was given. Twenty minutes later the patient was readily reversed with neostigmine 2.5 mg and atropine 1.2 mg. One litre of dextrose 4% and saline 0.18% was administered intra-operatively. The same measures that were used in case 3 to minimise heat loss were used. Emergence was uneventful and the patient had no muscle stiffness or weakness in the postoperative period.

Discussion

The anaesthetic management of these patients provided an interesting challenge because of the history of anaesthetic problems, the rarity of the condition and the limited literature available. An additional difficulty was presented by controversies about the classification of the periodic paralyses and their relationship to paramyotonia congenita. The members of this family clearly exhibit symptoms and signs of both periodic paralysis sensitive to potassium and myotonia that is exacerbated by cold. Both aspects of the condition need to be addressed and appropriate precautions taken.

The family's muscle disorder had been clearly defined in the past. This was very helpful, since the nature of potassium sensitivity in any patient with periodic paralysis influences anaesthetic management. This may be defined by history alone but provocative testing has been suggested [22] to define the disease in any particular patient. Such testing had been carried out on the propositus when he first presented to the neurologist. In provocative testing, a glucose load with or without insulin is given initially and in the hypokalaemic form of periodic paralysis this will precipitate weakness. If this test is negative, but an attack of weakness occurs in response to an oral potassium load, the hyperkalaemic form is diagnosed. Intravenous calcium gluconate had been given to the propositus whilst weak from the potassium load and this had aborted the attack. The value of this test had been reported in some patients with HPP by Gamstorp in 1956 [4] and van't Hoff in 1962 [6]. Calcium gluconate was kept at hand in the operating theatre in case it was required to decrease the serum potassium concentration.

Several other drugs have been reported as being of use in aborting an attack of weakness in HPP. These include insulin, glucagon, adrenaline, salbutamol and metaproterenol [14, 22–24]. However, not all drugs are effective in all patients.

Preparation for elective surgery should begin well in advance. In three out of the four patients, pre-operative potassium depletion with frusemide was undertaken. This was omitted for the third patient because of her long-term chlorothiazide therapy. Chlorothiazide and other thiazide diuretics have proved useful in the prevention of attacks in some patients with HPP [12, 21, 22] although they some-

times aggravate myotonic symptoms. Acetazolamide is also used to prevent paralytic attacks, presumably because of its kaliuretic effect [22]. However, not all patients respond and side effects may outweigh usefulness. Paradoxically, acetazolamide is also useful in decreasing frequency of attacks in the hypokalaemic form of periodic paralysis. The mechanism of action in this condition is less clear but it may in part relate to the production of a metabolic acidosis, which causes an influx of potassium into the intracellular compartment.

In the hypokalaemic form, ingestion of a large carbohydrate meal has been known to precipitate weakness. Conversely, in the hyperkalaemic form, starvation and carbohydrate depletion may cause weakness. In some families, missing a meal may cause weakness that can be reversed by eating [16]. To avoid carbohydrate depletion in the third and fourth patient a dextrose infusion was started as soon as the fasting period began and continued until oral intake resumed. The other two patients had dextrose infusions started either intra or postoperatively. The failure to begin infusion from the onset of fasting in two of the four patients did not cause a problem. However, a dextrose infusion should be provided if fasting is to be prolonged or if a history of weakness induced by starvation is obtained. Fluids containing potassium should be avoided because of the sensitivity to potassium in this disorder. However, the second patient received 500 ml of compound sodium lactate solution intra-operatively without causing problems, presumably because this volume contains less than 3 mmol of potassium in total.

Drugs which release potassium from cells should not be given and those which cause muscle hyperactivity, such as ketamine and prochlorperazine, should also be avoided. The use of suxamethonium is contraindicated not only for its hyperkalaemic effect, but because it may produce severe myotonia, and this occurred in two of these patients before the diagnosis was made. The myotonic muscle rigidity with suxamethonium could be mistaken for that of malignant hyperthermia (MH). However, there is no evidence of an association between HPP and MH. Lehmann-Horn and Iaizzo [25] recently reported *in vitro* contracture tests on muscles from patients with myotonia or periodic paralysis and concluded that there is no increased susceptibility to MH. In addition, none of our patients showed signs of MH, apart from muscle stiffness in response to suxamethonium, despite prolonged exposure to known trigger agents.

The presence of severe gastro-oesophageal reflux would normally have made suxamethonium the relaxant of choice in the third patient for rapidly securing the airway. Instead, we chose to decrease gastric acidity with ranitidine, pre-oxygenate, induce anaesthesia and apply cricoid pressure whilst performing gentle IPPV via a mask. Cricoid pressure was maintained until the airway was secured. An alternative strategy would have been to have performed an awake intubation. It has been suggested that all myoneural relaxants be avoided in patients with periodic paralysis because of undue sensitivity to their effects [18]. This advice had been followed in earlier anaesthetics given to the third patient at another hospital. However, there is no clear evidence that the presence of periodic paralysis results in increased sensitivity to nondepolarising agents. The use of deep inhalational anaesthesia sufficient to achieve relaxation may promote cooling, which itself is known to precipitate an attack [19]. The uneventful use of vecuronium in

a patient with HPP was reported in 1989 [19]. The four cases we describe all received nondepolarising myoneural relaxants in conventional doses. No sensitivity was demonstrated using neuromuscular monitoring and reversal was achieved at the end of the procedure.

Cold is an important trigger factor of both myotonia and weakness in this condition. Warming blankets were used and all intravenous fluids were warmed intra-operatively. Low fresh gas flows were used and a heat and moisture exchanger was included in the breathing circuit for the third and fourth patients to reduce the respiratory heat loss. In addition, for these two patients, legs and head were wrapped in plastic to further reduce heat loss. Oesophageal temperature was monitored in all cases and this remained within one degree Celsius of the initial temperature.

There were problems with bradycardia in case 3, which was probably related to the use of sympatholytic drugs in the presence of beta adrenergic blockade. Cardiac arrhythmias have been reported in a few patients with HPP [26, 27] but these consisted of paroxysmal tachycardias associated with episodes of weakness. Our patient had no pre-operative symptoms to suggest the occurrence of arrhythmias, but her pre-operative ECG showed a marked bradycardia therefore the possibility of sick sinus syndrome was considered. However, she responded to treatment with atropine and after a reduction in the dose of atenolol postoperatively, the patient maintained a sinus rhythm of 60 beat.min⁻¹.

The rôle of the type I anti-arrhythmic drugs in the management of myotonic symptoms has already been mentioned. The first patient found that procainamide therapy enabled him to undertake physical activity which his myotonia would have prevented. The efficacy of these drugs relates to their effect on sodium channel function. Tocainide has also been used, but the potential for bone marrow suppression precludes its use in a condition which is essentially benign [28]. Mexiletine has also been shown to be effective and preliminary studies suggest no long term adverse effects [29]. It may therefore have a future rôle in pre-operative prophylaxis for myotonic symptoms.

In summary, this paper describes a family affected by hyperkalaemic periodic paralysis with paramyotonia. Four adults had uneventful general anaesthetics using nondepolarising myoneural blockers for abdominal surgery, without the development of postoperative muscle weakness or myotonia.

On the basis of our experiences we make the following recommendations for anaesthesia in these patients: (a) pre-operative potassium depletion with diuretics; (b) prevention of carbohydrate depletion during fasting with intravenous dextrose solutions; (c) avoidance of potassium containing fluids and potassium releasing anaesthetic agents; (d) temperature monitoring and maintenance of normothermia; (e) continuous ECG monitoring and frequent serum electrolyte estimations; (f) the availability of calcium gluconate in case of an increase in serum potassium which may be manifested by T wave changes on ECG; (g) prophylactic treatment with mexiletine may be considered; (h) avoidance of suxamethonium; (i) on the basis of our observations in this family, the response to nondepolarising neuromuscular relaxants may be normal. It is therefore suggested that their use is not contraindicated provided that neuromuscular monitoring is undertaken.

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CASE REPORT

Delayed profound respiratory depression after premedication with trimeprazine

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Summary

Delayed, profound respiratory depression occurred in a 4-year-old boy, who had been premedicated with trimeprazine 4 h after tonsillectomy. This is a rare, but potentially fatal idiosyncratic reaction.

Key words

Anaesthesia; paediatric.

Pharmacology; trimeprazine.

Complications; respiratory depression.

Trimeprazine tartate is a phenothiazine derivative commonly used in paediatric practice for premedication, sedation and relief of pruritis [1]. It has central sedative, antihistaminic, antiemetic and spasmolytic actions. The recommended oral dose at the time of the present case was 3 mg.kg^{-1} [2,3]. The oral preparation (Vallergan Forte 6 mg.ml^{-1}) is administered as a syrup 1 h before operation [4]. Side effects can include dryness of mouth, vertigo, fainting and rarely agranulocytosis. Central nervous system depression and hypothermia may occur following an acute overdose.

A case of severe delayed respiratory depression requiring ventilatory support after premedication with trimeprazine is described.

Case history

A 4-year-old, 20 kg boy with a history of recurrent sore throat was admitted for elective tonsillectomy. There was no evidence of sleep or obstructive apnoea, he had had no previous general anaesthetics and past medical and family history were normal. Physical examination revealed no abnormality, he was taking no medication and had no known drug allergies.

He received trimeprazine syrup (60 mg) 1 h before operation. On arrival in the operating theatre he was drowsy but otherwise normal with a blood pressure of $100/50 \text{ mmHg}$, heart rate 120 min^{-1} and an oxygen saturation of 96%. Anaesthesia was induced with thiopentone

(5 mg.kg^{-1}), and following suxamethonium (1 mg.kg^{-1}) the patient's trachea was intubated with a size 5 Rae uncuffed tracheal tube. Anaesthesia was maintained with oxygen 30%, nitrous oxide 70% and isoflurane 2% delivered via an Ayre's T piece. Intra-operative monitoring was by continuous ECG, non-invasive automatic blood pressure and pulse oximetry. The patient was haemodynamically stable throughout the operation with a blood loss of 30 ml. The operation lasted 25 min during which time the patient received 150 ml intravenous solution.

The patient's trachea was extubated whilst he was on his side and he was transferred to the postoperative recovery ward. Recovery from anaesthesia was uneventful apart from restlessness, and intramuscular pethidine (15 mg) was administered for postoperative analgesia. After 30 min in the recovery area he was awake, obeying commands and maintaining a clear airway; he was then transferred to the ward. Four hours later it was observed that the child's breathing was laboured and he appeared cyanosed. An anaesthetist was called and on examination the patient was comatose and responded only to painful stimuli. Central cyanosis was present with a Cheyne Stoke's type respiratory pattern at a rate of $5 \text{ breath.min}^{-1}$; apnoeic periods lasted up to 20 s. The chest was clear to auscultation and there was no evidence of respiratory obstruction. A distinctive circumoral pallor surrounded by an erythematous type facial flush was noted. Both pupils were moderately constricted. The peripheries were well perfused with a blood pressure of $120/80 \text{ mmHg}$, a pulse rate of

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160 beat.min⁻¹ and a body temperature of 37.1 °C. Naloxone was administered intravenously in incremental doses, to a total of 200 µg with no effect.

The patient's trachea was re-intubated and the lungs ventilated with 100% oxygen and he was transferred to the Intensive Care Unit. IPPV was started at a rate of 15 breath.min⁻¹ with a tidal volume of 200 ml. On laryngoscopy there was no evidence of bleeding or tissue swelling and the vocal cords appeared normal.

Arterial blood gas analysis immediately after intubation showed a respiratory acidosis and gross hypercarbia (pH = 7.161, P_{O_2} = 33.43 kPa, P_{CO_2} = 10.13 kPa, base excess = +3.1 mmol.l⁻¹, actual bicarbonate = 26.1 mmol.l⁻¹, standard bicarbonate = 22.0 mmol.l⁻¹) which was corrected over the next hour by ventilatory support.

The chest X ray was normal. A full blood count, serum urea and electrolytes and blood glucose were all within normal limits. The child gradually began to improve and was sufficiently alert 6 h after admission to the Intensive Care Unit for the breathing system to be changed to a T-piece, on which he maintained satisfactory ventilation and arterial blood gases allowing his trachea to be extubated 1 h later.

He still remained drowsy and was observed in the Intensive Care Unit for a further 24 h before transfer to the ward where he made an uneventful recovery.

He underwent a right orchidopexy 6 months later with the omission of trimeprazine as a premedicant; anaesthesia and surgery were uneventful. He received 2.5 mg of intravenous morphine during operation for postoperative analgesia with no evidence of postoperative respiratory depression.

Discussion

Trimeprazine has been widely used as a paediatric premedicant [1], but there have been previous reports of cardiovascular and respiratory side effects. Mann [5] reported two cases of respiratory depression occurring in siblings after they received trimeprazine within the recommended dose range. The first case involved a 3-month-old, 6.3 kg boy who, 48 h after operation, received an oral dose of trimeprazine (2.4 mg.kg⁻¹) for night sedation. He had a respiratory arrest 5.5 h later and required resuscitation using oxygen via a facial mask and skin stimulation. He had a further respiratory arrest 9 h later and again responded to similar resuscitative measures. No other reasons for the respiratory arrests were found and he made a full recovery without any neurological or respiratory sequelae. His 3-year-old, 12 kg sister was given trimeprazine (2.9 mg.kg⁻¹) orally 5 days after operation before removal of sutures. Four hours later she became drowsy, and then deeply unconscious. She made a full recovery over the next 6 h.

Loan and Cuthbert [6] reported four cases in which the use of trimeprazine as a premedicant was associated with disturbing cardiorespiratory instability. The four children received doses ranging from 2.7 mg.kg⁻¹ to 4.2 mg.kg⁻¹, with an onset of symptoms and signs occurring within 80 min.

Surgery was deferred and they all made an uneventful recovery. It is important to note that all the cases had

suffered either recent respiratory tract infections or fever. Kahn and Blum [7] have suggested that trimeprazine in normal doses (1 mg.kg⁻¹ day⁻¹) could have been responsible for the deaths of seven infants with sudden infant death syndrome. In the period May 1967 to January 1990 the Committee for the Safety of Medicines in the United Kingdom has received two reports, both associating trimeprazine with respiratory arrest and dyspnoea (personal communication from the manufacturers, Rhone Poulenc Rorer Ltd).

The present case documents the delayed onset and profound respiratory depression that can occur following premedication with trimeprazine at a dose of 3 mg.kg⁻¹. The sequence of events was similar to those in the two cases reported by Mann [5], in that the onset of the respiratory depression did not take effect until approximately 6 h after administration of trimeprazine.

Although intramuscular pethidine (15 mg) was administered in recovery this was unlikely to be responsible for the respiratory symptoms as the patient did not respond to naloxone. He presented for right orchidopexy 6 months later and was given intravenous morphine (2.5 mg) with no side effects. At laryngoscopy there was no evidence of tonsillar bleeding, the vocal cords appeared normal and the patient was re-intubated with a size 5 uncuffed tracheal tube without any difficulties.

In this hospital all drugs given by the nurses are doubly checked and the likelihood of the patient receiving an overdose of trimeprazine would be remote. The fact that respiratory and central nervous system depression occurred on the ward after uneventful anaesthesia and recovery in the operating theatre is of particular concern, as such events, if left undetected, could result in a fatality. The severe nature of the respiratory depression, as supported by the clinical picture and arterial blood gases, and, which necessitated full ventilatory support for 6 h, also gives cause for concern.

The manufacturing company, has, as a result of the adverse side effects reported with the use of trimeprazine as a premedicant, reduced the recommended dose from 2 to 4 mg.kg⁻¹ to 2 mg.kg⁻¹ (personal correspondence).

This case report and literature review highlights a rare and potentially fatal idiosyncratic reaction to trimeprazine. We feel that the routine use of trimeprazine as a paediatric premedicant should be re-evaluated.

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CASE REPORT

Carbon dioxide pneumothorax occurring during laparoscopic cholecystectomy

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Summary

A previously fit patient underwent laparoscopic cholecystectomy. During the procedure arterial oxygen saturation fell and clinical examination revealed signs of a right pneumothorax confirmed by chest X ray. Aspiration of the pleural cavity and analysis of the gas removed showed it to be composed entirely of carbon dioxide. Possible mechanisms of entry of carbon dioxide into the pleural space are discussed.

Key words

*Complications; pneumothorax.
Surgery; laparoscopy, cholecystectomy.*

Laparoscopy is a well established surgical practice for diagnosis and minor surgical treatment. Recently the technique has been used for more major surgical procedures and with this may come new and important hazards. We report a serious complication occurring during laparoscopic cholecystectomy and discuss possible mechanisms for its development.

Case history

A previously fit 61-year-old woman who weighed 63 kg, presented for laparoscopic cholecystectomy. Anaesthesia was induced with propofol and alfentanil and muscle relaxation was provided by vecuronium. The trachea was intubated and auscultation revealed good bilateral air entry. Anaesthesia was maintained using nitrous oxide, oxygen and isoflurane delivered by intermittent positive pressure ventilation using a Manley Pulmovent. A tidal volume of 600 ml was used with a peak inspiratory airway pressure of 17 cmH₂O. Monitoring of the electrocardiogram, blood pressure (noninvasive), arterial oxygen saturation, end-tidal carbon dioxide, inspired oxygen concentration, expired tidal volume and airway pressures were used. The patient was placed in a 15° head-up tilt and four abdominal incisions were made: umbilical, epigastric and two others, 2 cm below the right costal margin in the mid-clavicular and anterior axillary lines. Carbon dioxide

was insufflated and the pneumoperitoneum maintained via the umbilical incision. Towards the end of the procedure (after approximately 50 min) the laparoscope and carbon dioxide insufflator were repositioned to allow removal of the gall bladder through the umbilical incision. Carbon dioxide now entered the peritoneal cavity through the epigastric incision. Shortly after this, the arterial oxygen saturation fell rapidly from 97% to 85%. All the monitored variables were unchanged (including the airway pressure), but clinical examination of the patient revealed a decreased air entry on the right side of the chest with a hyper-resonant percussion note. A chest X ray confirmed the clinical diagnosis of a right pneumothorax, occupying approximately half the hemithorax. A 14 gauge plastic cannula was inserted into the second intercostal space, midaxillary line and a total of 800 ml of gas aspirated. Analysis of this aspirated gas (Datex Cardiocap 2) revealed that it consisted of 100% carbon dioxide. Oxygen saturation increased to 96% with the evacuation of the pleural space and by this time surgery was finished. Neuromuscular block was reversed with neostigmine and glycopyrronium and the patient allowed to wake up. In view of the nature of the aspirated gas no further measures were taken. No subsequent respiratory problems ensued and a repeat chest X ray the next morning showed full re-expansion of the right lung. The patient was discharged home the following day.

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Discussion

Many complications of laparoscopy have been documented [1] and relate either to traumatic insertion of the laparoscope or to the effect of misplaced insufflating gas. Removal of the gall bladder via the laparoscope is a new but increasingly popular technique [2]. Hazards of the procedure are the same as those of any laparoscopy, but the location of the surgery and more invasive technique used, render it more prone to specific complications, notably damage to the common bile duct with leakage of bile, arterial bleeding and hepatic damage [2]. We have described a case of pneumothorax occurring during laparoscopic cholecystectomy. Pneumothorax may occur during anaesthesia as a result of barotrauma, rupture of congenital lung bullae or iatrogenic needle insertion into the pleural cavity during attempted central venous cannulation. None of these mechanisms is applicable in this case, however, since the gas (carbon dioxide) aspirated from the pleural cavity must have originated in the peritoneal cavity.

There are a number of possible routes by which carbon dioxide may enter the pleural space from its point of insertion into the peritoneal cavity [3–5]. These include surgical trauma to the diaphragm and pleura, a congenital pleuroperitoneal communication or a subperitoneal route. Passage of carbon dioxide from the peritoneal to pleural cavity through any of these routes would have been made more likely by the head-up position of the patient. A final possibility, unique to this procedure, is the passage of gas up through the falciform ligament which was perforated by the epigastric cannula during surgery. This ligament

supports the liver on the underside of the diaphragm and its two layers divide superiorly to leave a 'bare' area through which gas may theoretically enter the mediastinum via the caval orifice. The mediastinal pleura may subsequently rupture due to a build-up of pressure with a resultant pneumothorax. Whatever the actual mechanism, peri-operative diagnosis of the nature of the pleural space gas was useful since it enabled us to exclude a number of potential causes of pneumothorax. It also allowed us to predict that as the lung was not the source of the pleural gas, recurrence of the pneumothorax should not occur once insufflation of carbon dioxide had been discontinued. This report should make anaesthetists aware of the possibility of gas insufflated during laparoscopy producing a pneumothorax, particularly with the increased use of the technique for major upper abdominal procedures.

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CASE REPORT

Instrumental bronchial tears

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Summary

Two case reports of bronchial tears following airway instrumentation are presented, one of which resulted in death. Both patients developed pneumothoraces and other complications after attempts had been made under general anaesthesia to insert bronchial stents. It appeared that bronchial tears were made during instrumentation with the stent introducer and these cases demonstrate that great care should be taken when rigid materials, such as plastic guides and bougies, are used blindly in the airway.

Key words

Equipment; bougies, introducers.

Complications; airway, bronchial rupture, pneumothorax.

There is an increasing trend towards replacing procedures associated with airway management, traditionally performed under direct vision, with blind techniques; examples include the insertion of minitracheostomies, the changing of tracheal tubes and the fashioning of tracheostomies [1]. The equipment used for these procedures often includes rigid or semirigid lengths of plastic which at some stage are pushed into the trachea. The possibility and the consequences of perforating a bronchus during such instrumentation is illustrated by two patients in whom plastic introducers were used for the insertion of bronchial stents.

Case histories

Case 1

A 63-year-old woman presented with a bronchogenic carcinoma of her left main bronchus which extended onto the carina. The tumour had reduced the bronchial diameter to approximately 2 mm, and also partly obliterated the trachea. A course of carbon dioxide laser treatment under general anaesthesia was started. Partial clearance of the bronchus proved possible but it was judged that a better palliative result would be achieved if the area was supported with a stent. The plastic stent, mounted on a gum-elastic bougie, was pushed into position through a rigid bronchoscope. This manoeuvre was somewhat traumatic requiring several attempts before the stent was deemed to be in position. Some of the trauma and bleeding was related to difficulty in disengaging the stent from the bougie.

Shortly after waking, the patient developed severe respiratory distress and no relief was possible, even with her in a sitting position. Consequently she was re-anaesthetised and a bronchoscopy performed. The area where the stent had been placed proved difficult to visualise and it was decided to insert a tracheal tube in order to ventilate the patient's lungs. Following tracheal intubation, circulatory collapse occurred and it was also noted that the patient's abdomen was distending in time with ventilation. Needle aspiration of the abdomen led to a temporary improvement in the haemodynamic state. However, despite additional needle aspiration of both pleurae, the patient's condition rapidly deteriorated until cardiac arrest occurred, from which she could not be resuscitated. An antemortem chest X ray showed bilateral pneumothoraces, a pneumopericardium and a pneumoperitoneum.

Case 2

A 24-year-old man, who had previously had bilateral lung transplants for cystic fibrosis, was admitted because the right bronchial anastomosis had become narrowed and required insertion of a stent to ensure its patency. Under general anaesthesia a plastic stent, mounted on a bougie, was inserted. Some force was necessary but eventually the stent appeared to be in a satisfactory position. Respiratory distress was obvious as soon as the rigid bronchoscope was removed and neuromuscular blockade reversed. A tracheal tube was inserted and the lungs ventilated with 100% oxygen. A right pneumothorax was diagnosed clinically and needle aspiration of the right hemithorax produced an

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audible escape of air. A chest drain was inserted and the patient maintained on ventilation for the next 12 h. Extubation was uneventful and by 24 h the patient was fully awake and the chest drain had been removed.

Discussion

It has been the author's unfortunate experience, whilst inducing a patient for thoracic surgery, to push a gum-elastic bougie through the wall of a normal bronchus and create a tear that had to be sutured at the subsequent thoracotomy. Both the cases reported here were at even greater risk as they had significant airway pathology which might predispose to bronchial perforation.

The plastic and gum-elastic materials used in each of these cases are similar in shape and consistency to those in commercially marketed kits for procedures such as minitracheostomy, tracheal tube exchange, and percutaneous

tracheostomy. With a small degree of force these can perforate the membranous portions of the trachea and bronchi. The potential for tracheal tube stylets to cause similar damage has been highlighted previously [2].

Any sudden deterioration of a patient in whom such instrumentation has occurred should alert one to the possibility of a bronchial tear, and further deterioration in association with the application of positive pressure to the airway is likely to indicate the development of a pneumothorax. Speedy treatment of this possibility with needle aspiration may be lifesaving.

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An evaluation of oxygen delivery using nasal prongs

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Summary

Oxygen delivery using nasal prongs was assessed using a lung model for spontaneous ventilation. The analogue lung was attached to a manikin, which provided a model of the 'face and pharynx' to which the nasal prongs were applied. Oxygen concentrations were measured in the model trachea at varying fresh gas inflow and peak inspiratory flows. The study demonstrated enormous variability in the both the peak-inspired (26.3–90.0%) and end-expired concentrations (25.2–78.6%) of oxygen delivered to the trachea. There was a regular relationship between the ratio of peak inspiratory flows, expressed over fresh gas inflow and the end-expired oxygen concentrations which could allow estimation of inspired oxygen concentration.

Key words

Oxygen; delivery systems.

The administration of oxygen by nasal prongs is an old and simple technique which is still popular. It is suitable when prolonged oxygen treatment is required, particularly in the postoperative period. Recently, it has been demonstrated that oxygen by nasal prongs prevents episodic hypoxaemia (arterial oxygen saturation < 90%) during the night after upper abdominal surgery in normal patients [1]. However, the major disadvantage with this mode of oxygen delivery is that there is uncertainty about the inspired oxygen concentration. Leigh aptly regards the administration of oxygen under such circumstances as a therapeutic trial whose value would be infinitely greater if the inspired oxygen is always known [2].

Clinically, it is difficult to evaluate the range of inspired oxygen concentrations in humans, because of problems obtaining noninvasive inspired gas concentrations from accessible sites. The present study therefore used a lung model to investigate quantitatively the inspired oxygen administered by this route.

Method

Apparatus

A lung model driven by a modified Cape Waine ventilator was used to simulate spontaneous ventilation [3]. The waveform produced approximated to a sine wave. The patient-device interface was constructed using the face shield of a resuscitation manikin (Resusci Anne, Laerdal). This was attached by a tubing whose volume approximated

to the tracheal volume (130 ml), to the lung (Fig. 1). In this study tidal volume (V_t) was maintained constant at 400 ml and the peak inspiratory flow (PIF) was varied by altering the frequency of breaths.

Oxygen was delivered to the nasal prongs (Hudson Oxygen Therapy Ltd) using calibrated rotating bobbin flowmeters (Gap-Meter A₁₀ and A₆), the accuracy of which were checked using a dry rolling-seal spirometer (AirCo Ohio 840). PIF was measured using a Fleisch number 2 pneumatachograph. Gas concentrations were measured using a mass spectrometer (Centronics MGA), sampling at 30 ml.min⁻¹ via a fine bore catheter positioned in the opening of the 'trachea' of the model. The mass spectrometer was calibrated with a British Oxygen Company (BOC) certified gas mixture. The signals from this analyser were recorded continuously into a computer multi-channel chart program (MacLab, Analog Digital Instruments).

Measurements

Before the start of the experiment, the physiological dead space (V_d/V_t) of the lung model was determined using the modified Bohr's formula:

$$V_d/V_t = \frac{PE'CO_2 - PECO_2}{PE'CO_2}$$

where $PE'CO_2$ and $PECO_2$ are the peak and mean expired carbon dioxide (CO_2) partial pressures at equilibrium respectively.

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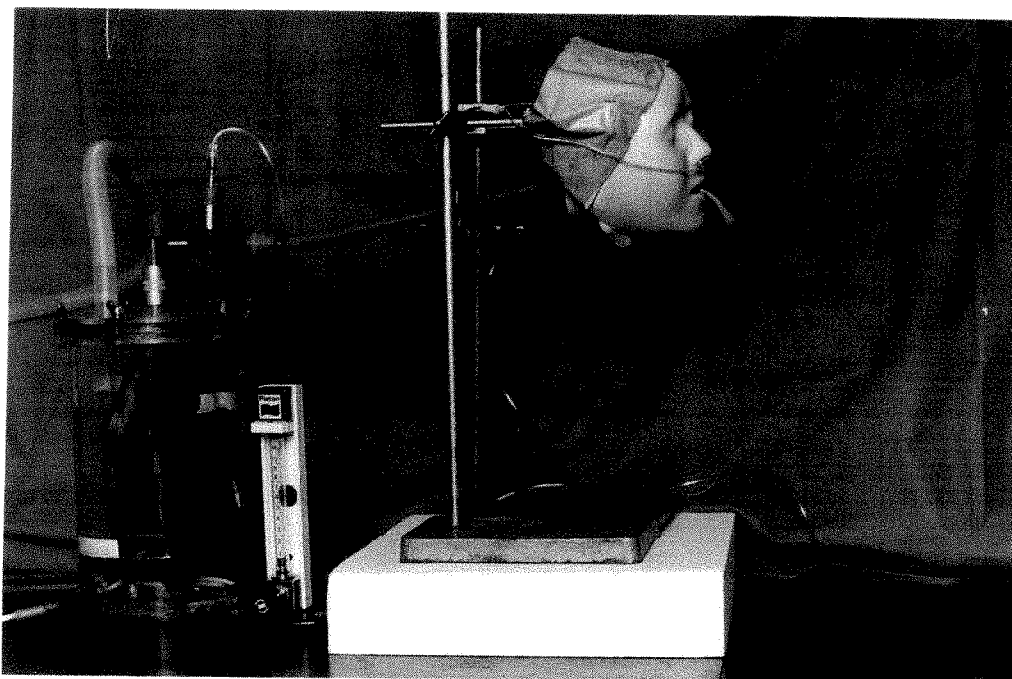


Fig. 1. The lung model with patient-device interface. The space between the bag and bottle acts as the lung and the bag is inflated cyclically by a modified Cape Wayne ventilator to produce a sine wave respiratory pattern.

In the study, the influence of PIF and fresh gas inflow ($\dot{V}_f$) on oxygen delivery were assessed independently. Oxygen measurements were obtained at equilibrium, which was taken to have occurred when the readings were steady over a 5 min period. The O_2 concentrations at various parts of the respiratory cycle i.e. peak-inspired (FO_{2peak}) and end-expired ($FE'O_2$) were determined from the recorded waveform. The individual parts of the waveform were identified by introducing CO_2 into the lung as a tracer gas. The CO_2 served as a tracer gas for the respiratory cycle.

Each step of this study was repeated on a separate day to check on the consistency of the measurements taken.

Results

The dead space (V_d/V_t) calculated by the modified Bohr equation for the lung model was 0.37, which was in close agreement with the value obtained from the ratio of their geometric volumes 0.33.

The values for oxygen concentrations did not differ by more than 0.8% between repeat measurements on separate occasions. Oxygen concentrations, when $\dot{V}_f$ and PIF were

altered independently, are represented in Table 1. The peak-inspired oxygen concentration values were higher than the end-expired values at all $\dot{V}_f$. The differences between the end-expired and peak-inspired O_2 values range between 0.3 to 11.4%, being more marked at higher $\dot{V}_f$ settings and lower PIF. Both oxygen values increase when $\dot{V}_f$ was increased. Conversely, the oxygen values decrease when PIF increase.

Discussion

The results of the study show that the effective inspired oxygen concentration increases with increasing $\dot{V}_f$ and varies inversely with PIF (Fig. 2). Low peak inspiratory flows can dramatically alter the oxygen delivered, particularly at high O_2 inflow rates. The study demonstrates the variability of O_2 concentration within a single inspiration; the end-expired value was taken to reflect the effective or mixed-inspired concentration. This is because in the lung model, where there is no O_2 uptake and CO_2 excretion, the effective oxygen concentration will be given by the alveolar concentration which will equilibrate with the end-expired

Table 1. Oxygen concentrations delivered to trachea of lung model.

$\dot{V}_f$ (l.min ⁻¹)	PIF = 12 (l.min ⁻¹)		PIF = 20 (l.min ⁻¹)		PIF = 30 (l.min ⁻¹)		PIF = 40 (l.min ⁻¹)	
	FO_{2peak} (%)	$FE'O_2$ (%)	FO_{2peak} (%)	$FE'O_2$ (%)	FO_{2peak} (%)	$FE'O_2$ (%)	FO_{2peak} (%)	$FE'O_2$ (%)
1	38.4	31.4	31.4	26.3	28.6	25.9	26.3	25.2
2	52.0	41.6	43.9	34.5	36.9	32.6	32.6	29.8
3	61.4	49.0	49.8	41.2	41.5	36.8	36.8	33.4
4	70.0	57.5	56.3	46.2	47.4	41.6	41.2	38.0
6	83.2	69.7	65.3	56.4	56.3	50.2	49.3	44.7
8	90.0	78.6	74.0	64.6	57.0	57.1	56.0	50.9

$\dot{V}_f$ = oxygen flow rate setting; PIF = peak inspiratory flow; FO_{2peak} = peak inspired oxygen concentration; $FE'O_2$ = end-expired oxygen concentration.

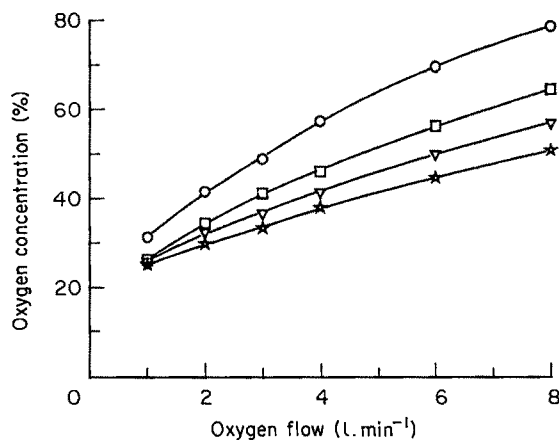


Fig. 2. Relationship of end-expired oxygen concentration to oxygen gas flow rate at various peak inspiratory flows (PIF). Symbols denoting PIF (l.min^{-1}): open box = 12; closed box = 20; triangle = 30 and hexagon = 40.

concentration. These results agree with the qualitative findings of Leigh [2, 4].

The use of nasal prongs was first described by Addis in 1963 [5]. There are several obvious clinical advantages to this system of oxygen delivery. These include easy handling, minimal interference with speaking, eating and drinking, good toleration by restless and comatose patients, and absence of 'suffocating' sensation associated with face-masks. The disadvantages have also been apparent; the most significant being the discrepancy between the inspired and delivered oxygen concentration. The size of the difference has been ascribed to inter-patient variables such as changes in minute ventilation, frequency of breaths, tidal volume, inspiratory time, expiratory time, functional apparatus dead space, inspiratory flow rate and expiratory flow rate in a wide variety of combinations.

Nasal prongs do not have a reservoir capacity, and consequently, neither apparatus dead space nor expiratory time should influence the delivered oxygen concentration. Leigh [2], in his comprehensive study of oxygen therapy devices, categorised them as a variable-performance system without a reservoir capacity. Oxygen delivery is therefore a function of peak inspiratory flow and oxygen flow rates [2, 4, 6].

This study demonstrates the relationship between PIF and $\dot{V}_f$. This can be used to determine the inspired O_2 concentrations by plotting O_2 concentration as a function of the ratio $\text{PIF}/\dot{V}_f$ (Fig. 3). The resultant plot has an exponential asymptote function appearance. Applying a fitted exponential curve, the graph can be applied clinically to predict the likely inspired oxygen fraction. $\dot{V}_f$ is easily measured and an estimate of PIF can be made, given that the resting PIF of adults ranges between 20 and

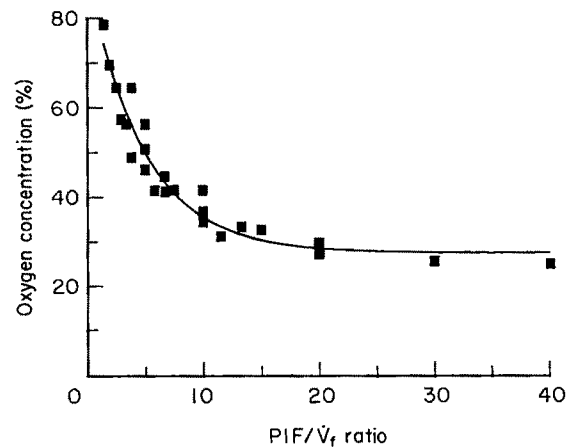


Fig. 3. The relationship between end-expired oxygen concentration and the ratio of peak inspiratory flow expressed over oxygen flow ($\text{PIF}/\dot{V}_f$). Graph constructed using fitted equation: $Y = 90.63 * (1 - e^{-0.2x})$.

30 l.min^{-1} [7]. This provides the prescriber with some degree of manipulation of the inspired oxygen fraction of the patient.

The current management of oxygen therapy is by the measurement of patient end-points, such as arterial oxygen saturation (oximetry) or arterial blood gas tensions. The technique described here cannot supplant these measurements. As with all other drug therapy, a knowledge of the dose should be a fundamental part of the treatment. This, to date, using nasal prongs has been impossible. The data described in this study may be useful in giving some guidelines to the otherwise totally arbitrary gas flow settings used in clinical practice.

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A pressure regulator for the cuff of a tracheal tube

D. M. MILLER

Summary

A regulator is described for the maintenance of a constant pressure difference above airway pressure during positive pressure ventilation in a cuffed tracheal tube. It comprises a tubular threshold valve which is powered by the anaesthetic gas supply source to a breathing system. The valve is interposed between the anaesthetic gas supply machine and the breathing system creating a pressure differential. The upstream pressure is transmitted to the pilot tube supplying the cuff of a tracheal tube via an upstream connection. The valve is suitable only for breathing systems that require a constant gas supply. The regulator was evaluated during anaesthesia, using a modified Mallinkrodt Hi-Lo jet ventilation tube to obtain simultaneous pressure measurements within the cuff and the lumen of the tracheal tube. A greater pressure was demonstrated in the cuff than in the airway and the two traces were approximately parallel throughout the respiratory cycle. The device should prevent excessive cuff inflation pressure and solves the problem of forgetting to let the cuff down before extubation.

Key words

Equipment; pressure regulator, cuffed tracheal tube.

In order to prevent gas escaping past a tracheal tube, a relatively large volume cuff is inflated with air so that it forms an airtight seal with the trachea. The cuff also anchors the tracheal tube within the trachea below the vocal cords and prevents aspiration of pharyngeal contents.

These benefits require that the cuff is inflated to a pressure greater than airway pressure, a practice that frequently results in much higher pressures than the peak inflation pressures used for ventilation [1–3]. One study [3] gave a mean value for cuff pressures in males as high as 11.9 kPa and 13.5 kPa in females. Excessive cuff pressures may affect perfusion of the mucosal lining of the trachea and cause damage [4, 5]. The higher the pressures used, the poorer the perfusion that results and the greater the risk.

While many techniques for minimising these complications have been described [3, 5, 6], it would be of value to have a system that automatically ensures that the lowest cuff pressures necessary are used to achieve the sealing of the tracheal tube and prevention of aspiration. The ideal would automatically hold the cuff pressure just above airway pressure and greater than the maximum hydrostatic pressure which may develop from fluid in the pharynx (about 0.8 kPa). A device which allows variation in airway pressure to be transmitted to the cuff, and a minimum cuff pressure at about 0.8 to 1.0 kPa, would be desirable.

A number of patented designs have been described [7, 8] which vary in complexity. However, they have dis-

advantages, such as a fixed capacity [7], which may not compensate adequately for nitrous oxide diffusion, or complexity and the requirement of an external high pressure gas supply [8].

The simple apparatus described here is designed to provide safe, reliable, automatic control and maintenance of tracheal tube cuff pressure by a preset level above airway pressure during positive pressure ventilation. This is achieved without an extra gas supply.

Description of the regulator

A pressure regulator is described, (Fig. 1), which is powered by the anaesthetic gas supply to a breathing system. It maintains a constant pressure difference in a high volume, low pressure, cuffed tracheal tube above airway pressure during both positive pressure ventilation and spontaneous ventilation. The apparatus comprises a tubular threshold valve which, interposed between the anaesthetic gas supply machine and the breathing system, creates a pressure differential. The upstream pressure is transmitted to the pilot tube supplying the cuff of a tracheal tube. The valve is suitable only for breathing systems that require a constant gas supply.

The pressure differential created by the threshold valve may be either fixed or adjustable. The upstream portion of

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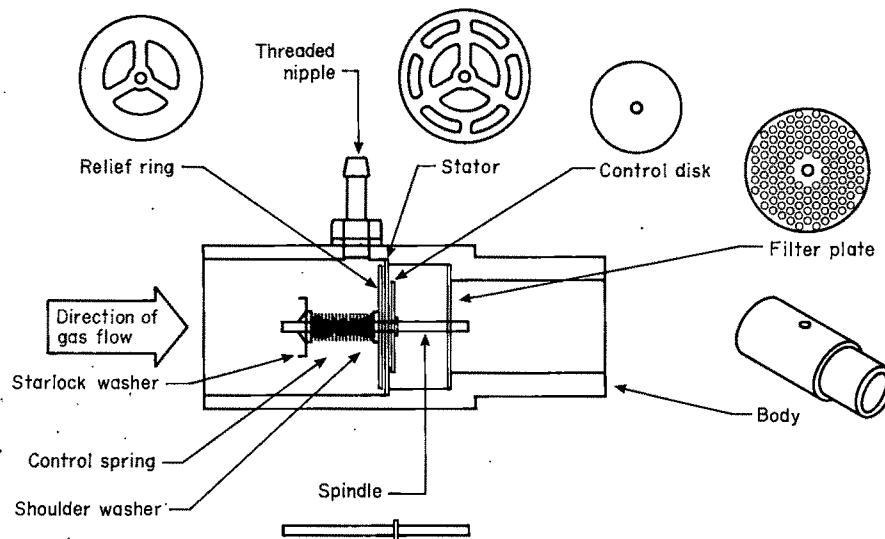


Fig. 1. A cross-section of a bidirectional flow cuff pressure regulator, indicating the direction of gas flow if the desired differential pressure is to be generated in the pilot tube connector, labelled threaded nipple.

the valve is in open communication with the cuff of a tracheal tube via the pilot tube connection. The purpose is to transmit and maintain the pressure in the cuff above that of the airway pressure by a constant value equal to the threshold valve setting.

In order to overcome the potential hazard of a one-way valve which may be incorrectly placed somewhere within a breathing system, the threshold valve is designed in such a way as to allow flow in both directions.

Methods

To find out whether cuff pressure was consistently maintained above airway pressure throughout the respiratory cycle, the simultaneous measurement of pressure within the cuff of the tracheal tube and within its lumen was recorded during various conditions of anaesthesia. These conditions included both spontaneous and controlled ventilation on two patients and the use of different fresh gas flows from 0.5 to 5 l.min⁻¹. Two disposable, high frequency, jet ventilation tubes, the Mallinkrodt Hi Lo, were modified for this purpose, as shown in Figure 2. Permission was granted from the medical ethics committee for this study and informed consent was obtained. The pressures within the cuff and the airway were measured and recorded using the

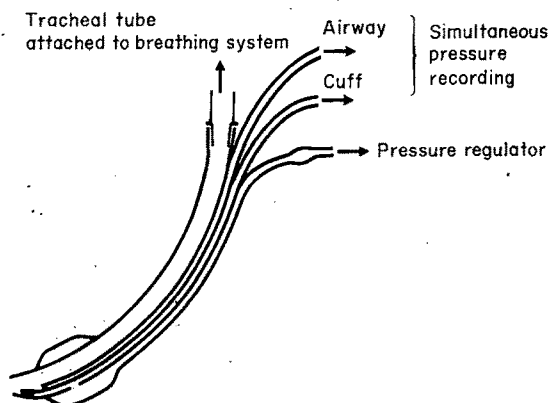


Fig. 2. This shows a cross-section of the modified Mallinkrodt Hi-Lo jet ventilation tube indicating the various connections.

following apparatus: Medex System MX 8014 with transducers (MX807) and domes (MX800) attached to a Sirecust 960 digital recorder and Siredoc 220 printer. The whole system and printed recordings were calibrated and checked against a water manometer.

Results

The results of the recordings are displayed in Figures 3 and 4. The influence of various oxygen flow rates through the valve upon the differential pressure generated was measured using a calibrated oxygen flow meter and a water manometer (Fig. 5).

Figure 3 demonstrates the pressures we currently aim to achieve in a normal tracheal cuff and tube. Figure 4 shows

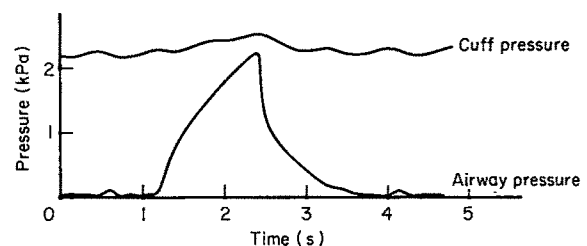


Fig. 3. An example of what is currently achieved using a normal cuff, with pressure monitoring.

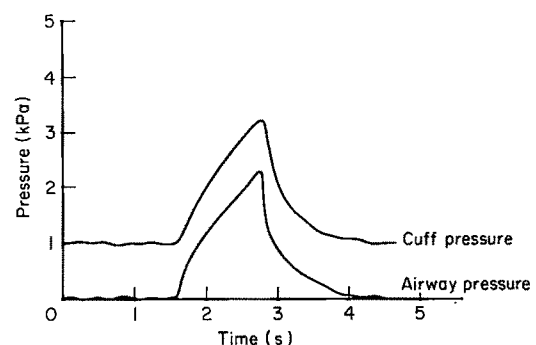


Fig. 4. This shows one typical trace obtained during positive pressure ventilation using the cuff pressure regulator with a fresh gas flow rate of 1 l.min⁻¹.

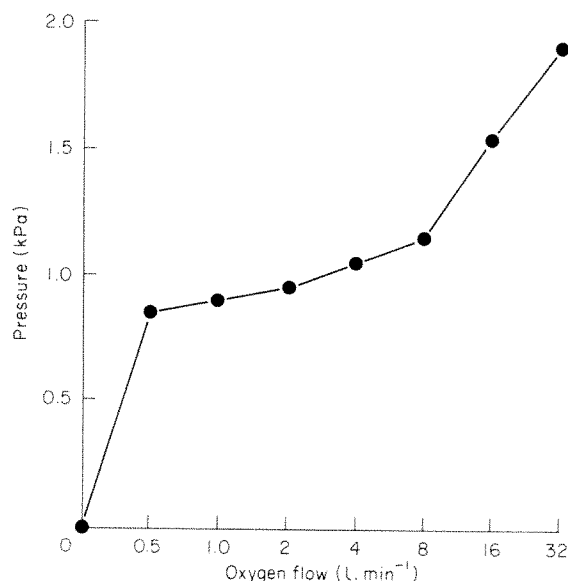


Fig. 5. This shows the typical pressure generated in the pilot tube of a cuff pressure regulator while using oxygen at different flows.

a typical trace obtained during positive pressure ventilation using a fresh gas flow [$\dot{V}_f$] of 1 L.min⁻¹. Here, cuff pressure is seen to be consistently greater than airway pressure throughout the respiratory cycle. With a $\dot{V}_f$ of 0.5 and 5.0 L.min⁻¹ respectively, traces almost identical in shape were obtained, but with pressure differentials of only 0.65 and 1.3 kPa. Figure 5 shows the resultant pressures above airway pressure that are generated by the valve using various oxygen flow rates. Figure 6 shows the position of the regulator whilst in use.

Discussion

The cuff pressure regulator is a simple device that is easy to use and its advantage to the patient is readily apparent. It overcomes all the mechanisms whereby cuff pressures of standard tracheal tubes can rise to dangerous levels.

The results demonstrate the effectiveness of the device at maintaining pressures appropriate to an adequate seal of the airway with $\dot{V}_f$ equal to or greater than 1 L.min⁻¹ in both spontaneous and controlled ventilation. A small delay in the rise of cuff pressure at the onset of inspiration was found as gas has to flow into the cuff in order to raise the pressure. The pressure differential was stable when $\dot{V}_f$ was equal to or greater than 1 L.min⁻¹ (Fig. 5). A minimum $\dot{V}_f$ of 1 L.min⁻¹ is therefore advisable. The use of emergency oxygen with a flow of 30 L.min⁻¹ results in a pressure difference of 1.7 to 2 kPa, which is still acceptable.

Another device [8], designed with the same objective as the one described here, requires a high pressure gas supply. One potential hazard of any device powered by an external gas source, is that the gas source may by some means transmit pressure to the cuff making a safety blow-off pressure release valve necessary. However, as there is no high pressure gas supply to this device, a blow-off valve is unnecessary. Minor cuff leaks may be compensated for by this device.

The problem of accidental extubation with an inflated cuff using this device is minimised or overcome as the cuff pressures are low and gas is free to escape from the cuff as it is pulled through the glottis.



Fig. 6. The regulator in use.

Acknowledgment

I am grateful to my colleague Dr K. Payne for reviewing the manuscript. Pressure regulators may be obtained from Miller Dent Ltd, The Magdalen Centre, Oxford Science Park, Oxford OX4 4GA. Patient application number GB 9018774-1.

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An evaluation of the Rapid Infusion System

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Summary

The performance of the Rapid Infusion System was evaluated in the laboratory. Using a conventional mixture of two units of packed red cells, two units of fresh frozen plasma and 500 ml crystalloid, a single line and a driving pressure of 300 mmHg, the highest flow in our study was 970 ml.min⁻¹ (2.8 mm catheter, no stopcock). With a 1.6 mm venous cannula the measured flow was 640 ml.min⁻¹. Additional diluting of the standard 'blood cocktail' did not add much to the performance of the system. When primed with tap water 21°C (12°C respectively), the fluid at the outlet of the system reached a maximum temperature of 37.8°C (37.4°C) after 6 min at a flow of 400 ml.min⁻¹. At flows higher than 1150 ml.min⁻¹ (priming with 12°C tap water: 800 ml.min⁻¹), the system slowed down to flows of 700 to 1000 ml.min⁻¹ in order to maintain an adequate temperature. We conclude, that the Rapid Infusion System is a valuable tool for situations where a rapid but controlled replacement with warmed blood at rates up to at least 1100 ml.min⁻¹ is needed. The use of large bore intravenous catheters and avoiding additional resistors such as standard 3-way stopcocks is highly recommended.

Key words

Equipment; blood warmer.

A rapid supply of adequately warmed fluids is the primary goal in the treatment of patients in hypovolaemic shock. Applying conventional methods of fluid replacement may result in inadequate warming, and consequently hypothermia, at high flows [1–6]. In addition, a system consisting of one or two pressure bags, a blood warmer and the interconnecting tubes may cause complications such as disconnections or difficulties in the measurement of the administered volume. The Rapid Infusion System (RIS) was developed in 1982 by J. Sassano at the Presbyterian University Hospital of Pittsburgh [7]. After a long development period, the final design of the machine was produced by Hemonetics Corporation (Braintree, MA, USA) towards the end of 1986. The system, which is now commercially available consists of a 3 l fluid reservoir with a 170 micron filter, a heat exchange unit, a roller pump, a 40 micron filter, an infusion rate monitor/controller and several safety mechanisms including three air detectors (Fig. 1). The RIS is one of three currently available products for rapid administration of warmed fluids [8–12]. The aim of this study was to test the heating capacity and flow generation of the system combined with various types of catheters and stopcocks.

Methods

Figure 2 shows the system and the various places where the pressure and temperature measurements were taken. For the flow measurements we used tap water or a mixture of two units of packed red cells, two units of fresh frozen plasma and 500 ml crystalloid. The RIS was assembled with various catheters and a standard (ID 1.4 mm) or a Hi-Flo 3-way stopcock (Medex Inc. Hilliard, Ohio, USA; ID 2.1 mm). The tips of the cannulae were located at 100 cm above the floor, that is 25 cm above the pressure measurement point in the RIS. They drained freely into a reservoir. Mean flows were determined by collecting the volume delivered by the system during periods of 30 s. Each measurement was repeated six times.

To analyse simultaneously the effects of the two different liquids and the various catheters on the relationship between flow and pressure, a multiple linear model was formulated. In order to linearise the relationship, the natural logarithm of flow and pressure, respectively, were taken.

The warming capacity was evaluated in a separate set of measurements. The reservoir of the system was filled with

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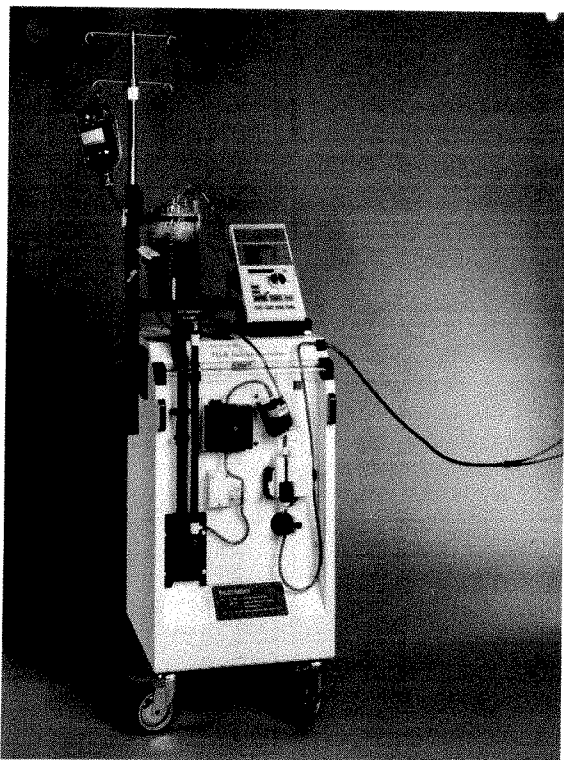


Fig. 1. The Rapid Infusion System.

tap water at 12°C and 21°C, respectively. Before each measurement, all tubes distal to the 40 micron filter were checked for equilibration to room temperature (21°C). The temperature of the water at the end of the tubes was then recorded with a NiCr-Ni thermocouple (Steiner+Co, Matzingen, Switzerland) at flows from 100 to 1500 ml.min⁻¹.

Results

Flows

When setting a driving pressure of 300 mmHg (40 kPa), using the 'blood cocktail' and a single line, the flows vary from 972 ml.min⁻¹ (1 Arrow-flex 8.5 French internal diameter (Fr ID) catheter, no stopcock) to 180 ml.min⁻¹ (using the side port of a Cook chek-flo II with a 7 Fr PA-Catheter in the main lumen) (Fig. 3). When using a venous cannula (Vigon 14 G outside diameter [OD], no stopcock) the measured flow is 641 ml.min⁻¹.

Analysis of the multiple linear model shows that there is a small but statistically significant difference of 9.4% (95% confidence interval: 7.7–11.0%) in the flows between tap water and the blood cocktail. The difference between the various catheter settings, however, is much more relevant, as shown by a 2.4-fold increase in the flow from a 17 G OD (1.15 mm ID) to a 14 G OD (1.60 mm ID), a 3.7-fold increase from a 17 G OD to a 12 G OD (2.15 mm ID) and a 5.7-fold increase from a 17 G OD to a 8.5 Fr ID (2.70 mm ID) cannula (Fig. 4, note the metric values for ID given in parenthesis were measured in our laboratory).

Warming capacity (Fig. 5)

When primed with tap water at 21°C (12°C respectively) and with a flow of 400 ml.min⁻¹, the fluid at the output of

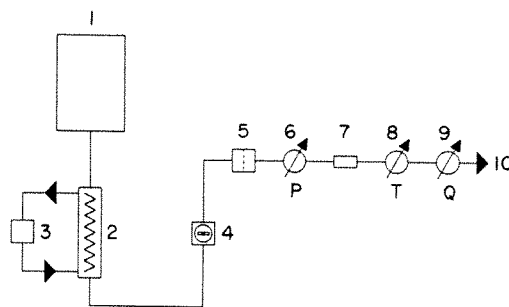


Fig. 2. Diagram of the Rapid Infusion System. 1, reservoir with 170 µm filter; 2, heat exchanger; 3, water reservoir (5 l); 4, roller pump; 5, blood filter 40 µm; 6, pressure monitor; 7, catheter, 3-way stopcock; 8, temperature monitor (Thermocouple); 9, flow measurement; 10, to patient.

the system reached 36.8°C (36.2°C) after 1 min. After another 5 min, the highest obtainable temperature of 37.8°C (37.4°C) was reached. At a flow higher than 1150 ml.min⁻¹ (800 ml.min⁻¹), the system slowed down to flow rates of 700 to 1000 ml.min⁻¹ because the output-temperature fell below 34°C after a few min.

Discussion

Hypothermia in trauma patients is often considered an ominous sign [13,14]. In other situations, such as extensive liver surgery and liver transplantation, massive transfusions may also be required, with the concomitant problem of hypothermia [15,16].

Russell reviewed blood warmers extensively in 1974 [17]. He recommended that the ideal warmer should be able to provide blood at a temperature greater than 32°C and at a flow above 150 ml.min⁻¹. Among the 11 items of equipment tested at that time, the Fenwal unit was the only one to meet these criteria. One of the major problems with blood warmers is that heat exchange through plastic materials is small compared with metal (thermal conductivity [18] (W.m⁻¹.°K⁻¹): copper 401, aluminium

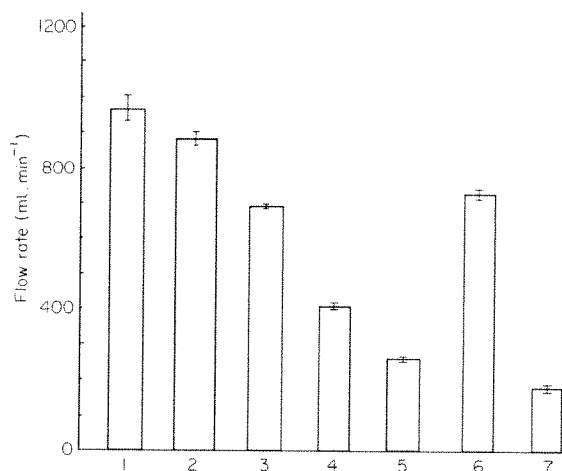


Fig. 3. Flows at 300 mmHg driving pressure with various catheters and 3-way stopcocks. All measurements are performed with one infusion line, including a 120 cm extension tube (mean SD). 1, Arrow 8.5 Fr; 2, Arrow 8.5 Fr, 2 stopc. large; 3, Arrow 8.5 Fr, 2 stopc. standard; 4, Arrow 8.5 Fr, 2 stopc. large, using side port; 5, same as 4 + PA catheter in main lumen; 6, Cook chek-flo II, 2 stopc. large, side port; 7, same as 6 + PA catheter in main lumen.

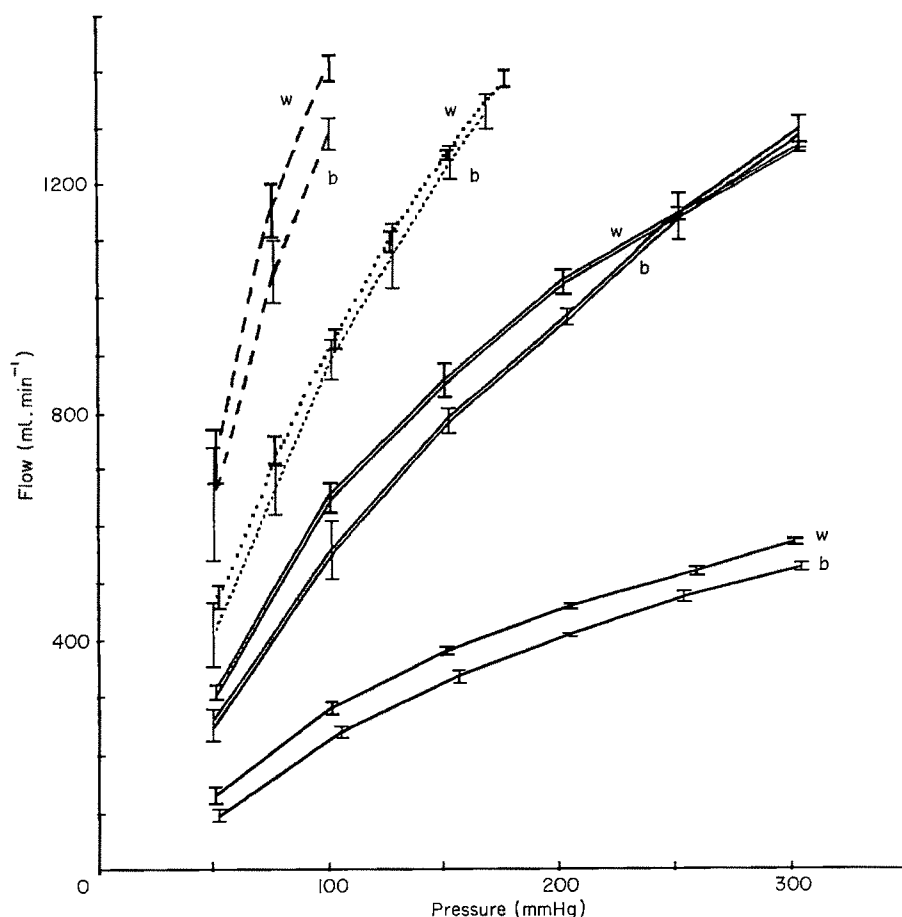


Fig. 4. Pressure vs. flow for various catheters, each measured with tap water(w) and blood cocktail(b). All measurements were with two infusion lines, including a 120 cm large-bore (3.3 mm ID) extension in both lines (mean SD). —, Venflon 17 G (1.15 mm ID); =, Venflon 14 G (1.6 mm ID);, Angiocath 12 G (2.15 mm ID); ---, Arrow 8.5 Fr (2.8 mm ID).

236, polyethylene about 0.35). Additionally, if the tubing of the warming system is too long, resistance increases significantly. Therefore, newer blood warming systems use a countercurrent heat exchanger with metal tubes which yield a good heat exchange.

In this study, we evaluated the performance of the Rapid Infusion System, which incorporates a single-channel coun-

tercurrent heat exchanger warmed to 39°C by water circulating from a 5 l reservoir. According to the specifications given by the manufacturer, the system allows flows up to 1500 mL.min⁻¹ with adequate warming of the fluids. We measured flow with several commonly available catheters. Additionally, two different 3-way stopcocks were used. Our measurements would probably vary slightly when using products from other manufacturers, since flows may vary between catheters of the same outer diameter but with other different characteristics [19]. In the clinical situation the occurrence of additional resistance (for example kinking of catheters [20]) has to be considered.

The side port of a pulmonary artery introducer catheter is sometimes used for transfusions. Benumof and workers [21] showed the flow through the side port of such catheters to be lower than for the same catheters without a side port. This was confirmed in our study. Furthermore the flow decreased considerably when a 7 Fr PA-catheter was added into the main lumen of large bore catheters, as shown in our investigation.

The flows for tap water and the 'blood cocktail' were similar, which is in accordance with the results of Floccare and workers [22]. With large bore tubing and catheters the effect of viscosity seems to be negligible and we found no clinically important differences.

Our measurements confirmed the maximal flow of 1500 mL.min⁻¹ for a combination system with two lines, each with two large-bore 3-way stopcocks, one large-bore

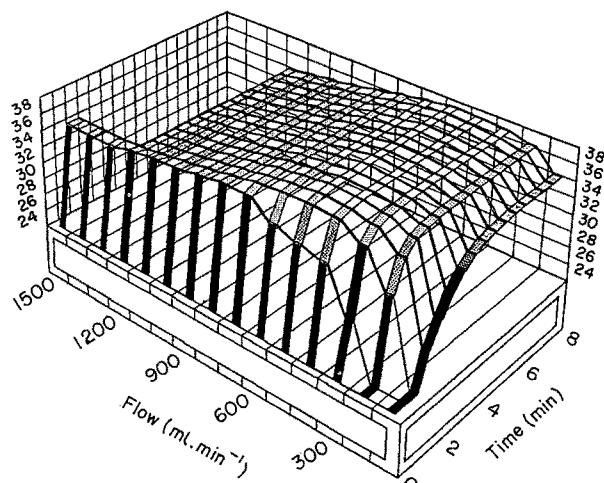


Fig. 5. Course of temperature (°C) recorded at the outlet of the system for tap water (21°C) and for various flows.

extension tube (120 cm long, 3.3 mm ID) and a 8.5 Fr Arrow-flex catheter, when using a 'blood cocktail' of two units of packed red cells, two units of fresh frozen plasma and 500 ml crystalloid. If such extremely high flow rates are used, the patient must be carefully monitored to avoid fluid overload.

We also tested the device for warming. Very high flows triggered a safety mechanism so that after a few minutes, as soon as the output temperature fell below 34°C, there was an automatic reduction in the flow rate. The limit was reached at flows of 1100 ml.min⁻¹ when using tap water of 21°C and at 800 ml.min⁻¹ with tap water of 12°C. It seems reasonable to have these limits. In clinical practice it will almost never be necessary to have such high flows for more than a few minutes.

At low flows (100 and 200 ml.min⁻¹) the temperature measured at the outlet of the system increased more slowly and a lower maximal temperature was reached because of the heat loss through the long interconnecting tubes [23] (Fig. 5). In clinical situations we therefore try to avoid the continuous use of such low flows. With relatively 'small' bleeds, we prefer to transfuse boluses of 500 ml. This can easily be done by pressing the respective button on the RIS.

The RIS allows for an easy and accurate dosage of volume. It is simple to operate, and leaves the anaesthetist's hands free for other tasks. Compared to other similar systems, however, the priming volume is quite large and the price for the hardware, as well as for the disposable sets, is high.

In conclusion, the RIS is a valuable tool for the controlled, rapid replacement of warmed blood at rates up to at least 1100 ml.min⁻¹. Additional dilution of the standard blood cocktail adds little to the performance of the system, while the use of large bore intravenous catheters and avoidance of additional resistors, such as standard 3-way stopcocks and side ports of introducers, is highly recommended.

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Forces applied during laryngoscopy and their relationship with patient characteristics

Influence of height, weight, age, sex and presence of maxillary incisors

M. J. L. BUCX, R. T. M. VAN GEEL, P. A. E. SCHECK, T. STIJNEN AND W. ERDMANN

Summary

The relationships between patients' height, weight, age, body mass index, gender and presence of maxillary incisors and a series of laryngoscopic factors have been studied. These included the duration of laryngoscopy, maximally applied force, mean applied force and the integral of force over time. There was a positive correlation between height and weight and laryngoscopic factors. Sex and age also showed a positive correlation but these could be explained by differences in height, weight and the presence of maxillary incisors. This latter factor was the dominant patient characteristic influencing the measured laryngoscopic factors. Use of these laryngoscopic factors as a measure of difficulty of laryngoscopy is discussed.

Key words

Anaesthesia, techniques; laryngoscopy.
Equipment; laryngoscope.

In a previous publication a curved blade laryngoscope was described which permitted the measurement of forces applied to the patient during laryngoscopy [1]. By means of this instrument four laryngoscopic factors can be quantified: (1) the duration of laryngoscopy (t_l), (2) the maximally applied force (F_{max}), (3) the mean force (F_{mean}) and (4) the integral of the forces over the time ($F \cdot t$). Quantification of these factors could be important when studying the relationship between laryngoscopy and intubation stimuli, and the resulting stress response [2]. Moreover, these factors might also be measured in studies concerning the difficulty of laryngoscopy or the effect of neuromuscular blocking drugs on intubation [3, 4]. In this context, the identification of patient characteristics which correlate with the laryngoscopic factors described above in 'routine' laryngoscopy and intubation is a logical first step. The purpose of this study was to investigate the relationship, if any, between these laryngoscopic factors and the patients' height, weight, body mass index, age, sex and the presence or absence of maxillary incisors.

Methods

Forty-nine patients, scheduled for elective surgery, participated in the study. Approval from the Hospital Ethics Committee and informed consent from each subject were obtained. All patients were ASA grade 1 or 2 adults, in

whom no difficulty with intubation was anticipated. Patients in whom tracheal intubation proved difficult, or was not achieved at first attempt, were excluded from the study. Before laryngoscopy the patient's head was placed in the 'sniffing' position. Laryngoscopy was performed by the same person on each occasion using the technique for curved blade laryngoscopes. In all patients, height (cm), weight (kg), sex, age and body mass index ($\text{kg} \cdot \text{m}^{-2}$) were recorded, as was the presence or absence of maxillary incisors.

The following laryngoscopic factors were measured: (1) the duration of laryngoscopy, (2) the maximally applied force, (3) the mean force and (4) the integral of the forces over the time. These were studied using a modified curved blade (Macintosh) laryngoscope as described previously [1]. This instrument measures forces acting along the axis of the handle of the laryngoscope by means of a strain gauge based sensor positioned between the handle and the blade. The instrument was calibrated before each measurement by placing a weight of 2 kg ($= 19.6 \text{ N}$) on to the laryngoscope blade, keeping the handle in the vertical position. Each patient received intramuscular midazolam 2.5 mg and intramuscular atropine 0.5 mg 60 min before surgery. Anaesthesia was induced with thiopentone $4\text{--}5 \text{ mg} \cdot \text{kg}^{-1}$, fentanyl $2 \mu\text{g} \cdot \text{kg}^{-1}$ and suxamethonium $1.0\text{--}1.5 \text{ mg} \cdot \text{kg}^{-1}$. Before intubation patients' lungs were manually ventilated with 66% nitrous oxide in oxygen.

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Statistical methods

To study the univariate relationships between the patient characteristics and the laryngoscopic factors, linear regression analysis or Student's two sample *t*-test was used. The result of the different groups were compared by the two-way unpaired Student's *t*-test. F^*t and t_1 were logarithmically transformed to obtain approximate normal distributions. To study the joint relationship of several patient characteristics simultaneously with the laryngoscopic factors, stepwise forward multiple linear regression analysis was performed. For each laryngoscopy factor, the best single discriminatory variable was selected; then the best pair of variables, which included the first, was determined and so on, until the addition of a further variable was no more significant. A *p* value of ≤ 0.05 was considered significant.

Results

The demographic details and the laryngoscopic factors studied are presented in Tables 1 and 2. There was a significant relationship between patients' height and F_{max} ($F_{max} = 0.5 \times \text{height} - 50$) ($p = 0.008$) ($r = 0.37$) and F^*t ($\log F^*t = 0.02 \times \text{height} - 1.63$) ($p = 0.01$) ($r = 0.37$). Furthermore, there was a significant relationship between weight and F_{max} ($F_{max} = 0.3 \times \text{weight} + 13$) ($p = 0.01$) ($r = 0.36$), F_{mean} ($F_{mean} = 0.2 \times \text{weight} + 6.7$) ($p = 0.004$) ($r = 0.40$) (Fig. 1.) and F^*t ($\log F^*t = 0.01 \times \text{weight} + 1.3$) ($p = 0.02$) ($r = 0.34$). The body mass index was significantly related to F_{mean} only ($F_{mean} = 0.5 \times \text{BMI} + 8$) ($p = 0.03$) ($r = 0.30$) and not to the other factors. A negative significant relationship was found between age and F_{max} ($F_{max} = -0.2 \times \text{age} + 45$) ($p = 0.02$) ($r = -0.34$).

All mean laryngoscopic measurements were greater in men than in women, but multiple regression analysis indicated that sex did not correlate with the laryngoscopic factors when corrected for height, weight and the presence of maxillary incisors. The presence of maxillary incisors had a significant influence on all laryngoscopic measurements (Table 3).

Table 1. Demographic details; values are mean (SD) and range, or ratios.

Height; cm	174 (9.0)	155–197
Weight; kg	74 (13.9)	50–113
Age; years	38 (17.0)	16–75
Body mass index; kg.m^{-2}	24.3 (3.8)	18.4–33.4
Male:Female	22 : 27	
With maxillary incisors:without maxillary incisors	40 : 9	

Table 2. Measured laryngoscopic factors.

	Mean	SD	Range
t_1 ; s	16.3	(11.8)	6.8–43.2
F_{max} ; N	35	(12)	13–60
F_{mean} ; N	20	(6)	9–32
F^*t ; N.s	324	(194)	66–981

t_1 duration of laryngoscopy; F_{max} , maximally applied force; F_{mean} , mean applied force; F^*t , force-time integral.

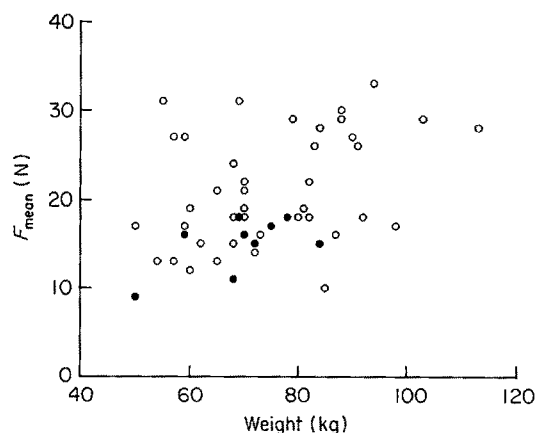


Fig. 1. Effect of weight (kg) on the mean applied force (N) during laryngoscopy. Patients with maxillary incisors (○) and without maxillary incisors (●) are indicated.

Stepwise forward regression analysis showed the presence of maxillary incisors to be the only variable with a statistically significant influence on the duration of laryngoscopy ($R^2 = 0.10$). For F_{max} the best single discriminatory variable was maxillary incisors ($R^2 = 0.27$); the best pair of variables was maxillary incisors and sex ($R^2 = 0.36$), the latter combining the effects of height and weight, making its predictive power greater than height or weight separately. Using the same method, for F_{mean} the best single variable was weight ($R^2 = 0.16$) (Fig. 1), the best pair of variables weight and maxillary incisors ($R^2 = 0.27$). For F^*t the best single variable was maxillary incisors ($R^2 = 0.21$) and the best pair of variables maxillary incisors and sex ($R^2 = 0.30$). The addition of a third variable did not significantly improve the correlations with laryngoscopic measurements. In all patients visualisation of the glottis was classed as grade 1 [5].

Discussion

Potentially there are many variables which might influence laryngoscopic measurements. These could be related to the patient, the person intubating (e.g. experience), and/or the anaesthetic technique e.g. the degree of muscle relaxation and the type of blade used. The forces applied during laryngoscopy to bring the glottis into view are those which are necessary to open the mandible, sometimes even to subluxate [6, 7], and to displace the tongue base/hyoid-complex forward.

Different criteria to predict the difficulty of laryngoscopy and intubation have been described [5, 8–10] assuming that mouth opening was sufficient to allow the introduction of

Table 3. The effect of presence or absence of maxillary incisors on the measured laryngoscopic factors; values are mean (SD).

Laryngoscopic factors	Present	Absent	<i>p</i>
t_1 ; s	17.4 (8.9)	11.4 (2.9)	0.001
F_{max} ; N	39 (11)	23 (5)	< 0.001
F_{mean} ; N	21 (6)	15 (3)	< 0.001
F^*t ; N.s	364 (201)	178 (70)	< 0.001

t_1 duration of laryngoscopy; F_{max} , maximally applied force; F_{mean} , mean applied force; F^*t , force-time integral.

the blade. The amount of atlanto-occipital extension has been identified as an important predictor of difficult laryngoscopy. Also important is the size of the tongue relative to the mandibular space available [5, 7, 8]. In turn, the volume of the mandibular space is related to the mobility and possibility of (sub) luxation of the temporomandibular joint, the adequacy of mouth opening and the ratio of the length of the mandible and maxilla. The concept of the line of vision [5, 7] explains the importance of the ratio of tongue size and mandibular space, bearing in mind that it is a construction in only two dimensions; the greater the proportion of tongue behind the line of vision, the more forceful will be the effort needed to displace the tongue base/hyoid-complex forward for visualisation of the glottis [7]. This implies, not surprisingly, that forces applied during laryngoscopy increase as the degree of difficulty increases, an association confirmed by several studies [11, 12]. In addition, both calcification of the stylohyoid ligament, disabling the forward displacement of the hyoid, and inadequate traction on the laryngoscope were identified as causes of difficult laryngoscopy [13, 14].

In this study the relationship between some easily identified patient characteristics and laryngoscopic factors measured during tracheal intubation were investigated. The fact that many different variables influence these factors is evident from the large standard deviations and small correlation coefficients. As expected, there was a positive correlation between laryngoscopic factors and the height and weight of the patients, but body mass index had only a minor influence. Age and sex were univariately related to laryngoscopic factors, but multivariate regression analysis proved these relationships could be explained by differences in height, weight and the presence of maxillary incisors. An independent effect of age on masticatory muscle weakening [15, 16] could not be detected in this study, but the presence of maxillary incisors had a strong influence on laryngoscopic measurements. A possible explanation for this might be that in the absence of maxillary incisors the part of the tongue behind the line of vision is smaller, reducing the effort needed to displace the tongue forward. In addition, resorption of the maxillary alveolus after loss of maxillary incisors is a contributing factor, although this phenomenon is more prominent in the mandible. Moreover, the loss of maxillary incisors weakens the muscles of mastication [15, 17].

The duration of laryngoscopy correlated only with the presence of maxillary incisors and this can be explained by the additional care required to prevent damage to the teeth.

In conclusion, the laryngoscopic factors studied here are related to some easily identified patient characteristics, the presence of maxillary incisors being the most important. These factors may be a useful guide to possible difficult laryngoscopy, and can supplement the grading system introduced by Cormack and Lehane [5].

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Forum

The minimum effective dose of lignocaine to prevent injection pain due to propofol in children

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Summary

In a single-blind study of 100 children aged 1 to 10 years, the minimum effective dose of lignocaine required to prevent injection pain due to propofol was 0.2 mg.kg^{-1} when veins on the dorsum of the hand were used. This is more than twice the adult value. We concluded that injection pain should not limit the use of propofol in children if an adequate amount of lignocaine is mixed immediately prior to injection.

Key words

Anaesthetics, intravenous; propofol.

Anaesthetics local; lignocaine.

Complications; pain

Injection pain is the commonest problem encountered when propofol is administered, occurring in up to 73% of adults [1], and 85% of children [2]. One successful strategy for pain prevention is the addition of lignocaine to propofol immediately before injection [3, 4]. In a group of 50 unpremedicated children, lignocaine (1 mg.kg^{-1}) mixed with propofol (3 mg.kg^{-1}) resulted in abolition of injection pain [5]. However, this dose of lignocaine was chosen empirically and the aim of the present study was to determine the minimum effective dose of lignocaine.

Methods

The study was approved by the hospital Ethics Committee and was carried out under the automatic exemption conditions of the Medicines Act 1965 (revised 1985) [6]. Informed consent was obtained from the parents of 100 children, 50 aged 1 to < 5 years and 50 aged 5 to < 10 years. Children with known sensitivity to local anaesthetics, egg protein, soya bean emulsion or a significant history of atopy were specifically not studied. No sedative or anticholinergic premedication was used but all children had EMLA cream applied to the dorsum of each hand at least 1 h before venous cannulation (22 gauge Venflon, Vygon UK).

Propofol 3 mg.kg^{-1} was drawn up into a syringe and lignocaine was mixed immediately before injection according to the following scheme: the first child in each age group received lignocaine 1 mg.kg^{-1} . If no pain on injection was observed then the next child received a dose of lignocaine reduced by 0.1 mg.kg^{-1} . This sequence was followed until pain was observed when the next patient received 0.1 mg.kg^{-1} increased dose of lignocaine.

Injection pain was assessed by an observer who was unaware of the lignocaine dose used. A four point beha-

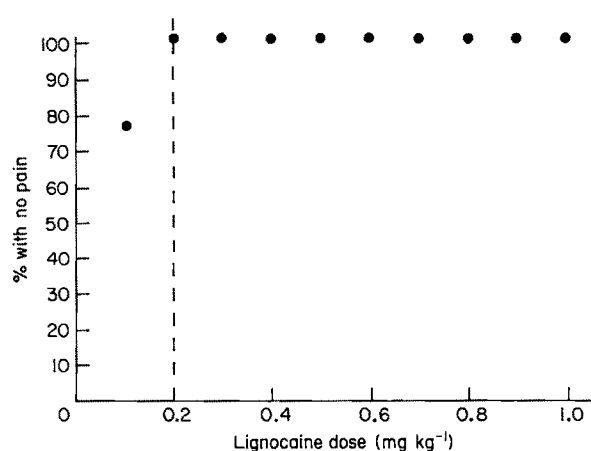


Fig. 1. Percentage of children aged 1 to 10 years where no pain was observed at each lignocaine dose level. No child who received 0.2 mg.kg^{-1} or more experienced pain. 22.5% of children who received 0.1 mg.kg^{-1} experienced pain.

vioural rating scale was used (no pain = 0; mild pain (grimace) = 1; moderate pain (grimace + cry) = 2; severe pain (cry + withdraw limb) = 3).

Results

One hundred children were studied. Their median age was 5.5 years (range 1.08–9.83) and their median weight was 22.7 kg (range 9.7–35.7). The proportion of children in whom no injection pain occurred is detailed for each dose level of lignocaine in Figure 1. No child was assessed as having injection pain who received 0.2 mg.kg^{-1} or more of

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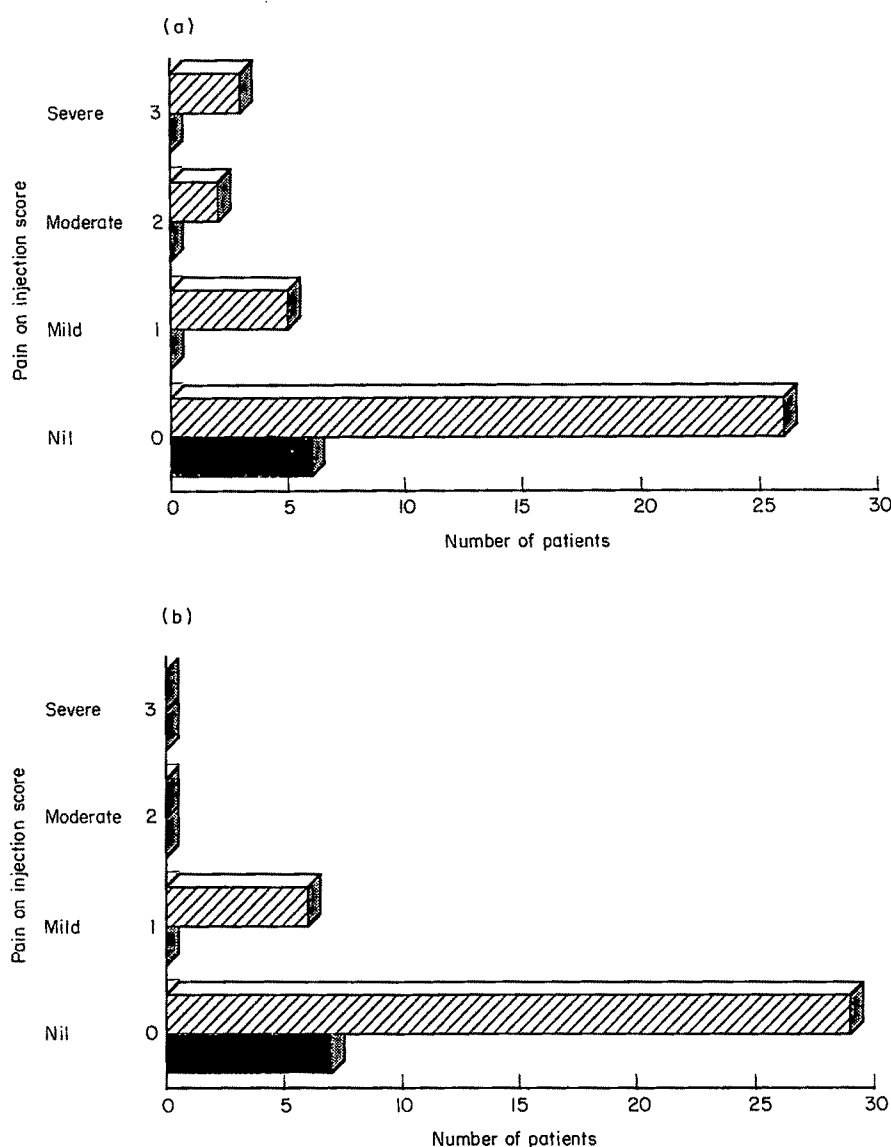


Fig. 2(a). Pain on injection scores for children aged 1 to < 5 years receiving 0.1 (▨) and 0.2 (■) mg.kg⁻¹ lignocaine (0 = no pain; 1 = mild pain; 2 = moderate pain; 3 = severe pain). (b) Pain on injection scores for children aged 5 to 10 years receiving 0.1 (▨) and 0.2 (■) mg.kg⁻¹ lignocaine (0 = no pain; 1 = mild pain; 2 = moderate pain; 3 = severe pain). If pain on injection was experienced, it tended to be more severe in younger children, aged 1 to < 5 years.

lignocaine. 22.5% of children who had received 0.1 mg.kg⁻¹ lignocaine were assessed as having injection pain. Pain was assessed as being moderate or severe in five patients in the 1 to < 5 year age group (Figs 2(a) and (b)).

Discussion

One method of preventing injection pain due to propofol in adults, is to use a large antecubital vein [4]. In children, this results in injection pain in 6.6% [7] to 24% [8]. However, the antecubital fossa carries the risk of arterial or neural damage and in children the availability of EMLA cream has made painless cannulation of veins on the dorsum of the hand the preferred choice of most anaesthetists.

This study confirmed the efficacy of added lignocaine in preventing injection pain due to propofol. However, an adequate dose must be used in children where 22.5% of 1 to 10 year olds experienced pain despite the addition of 0.1 mg.kg⁻¹ lignocaine to 3 mg.kg⁻¹ propofol. In younger children aged 1 to 5 years, injection pain tends to be more severe. In adults, the minimum effective dose of lignocaine

to prevent injection pain is 0.08 mg.kg⁻¹ (0.025%) [9], whereas our study indicates that for children aged 1 to 10 years, 0.2 mg.kg⁻¹ is required. Thus, children's veins would appear to be twice as susceptible to this adverse effect of propofol.

Previous studies in children have used an adaptation of the recommended adult regimen of 10 mg lignocaine per 200 mg propofol [10, 11]. At an induction dose of 3 mg.kg⁻¹ this produces a lignocaine dose of 0.15 mg.kg⁻¹. However, these studies found an incidence of injection pain of 29% and 60% respectively. Efficacy of added lignocaine is known to decline after 30 min [12] and it is possible that mixing immediately before administration in the present study was responsible for the efficacy of 0.2 mg.kg⁻¹. Any delay between mixing lignocaine with propofol and administration may result in a critical reduction in the concentration of lignocaine in the nonlipid phase of the emulsion, i.e. in the amount available to block afferent nerve fibres in the vein wall [13].

We conclude that the minimum effective dose of lignocaine required to prevent injection pain due to propofol

3 mg.kg⁻¹ in children is 0.2 mg.kg⁻¹. Injection pain is not a problem in children who receive propofol if an adequate amount of lignocaine is mixed with the propofol immediately before administration.

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Continuous paravertebral block in children

Initial experience

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Summary

Continuous paravertebral block has not previously been described for use in children. This study reports the use of this technique in five patients (aged 7 months to 8 years) scheduled for renal surgery or cholecystectomy. The block was not difficult to perform and provided good intra- and postoperative analgesia. Continuous paravertebral block appears to be a promising technique for pain management in children and might prove a valuable addition in the field of paediatric regional anaesthesia.

Key words

*Anaesthetic techniques, regional; paravertebral block.
Anaesthesia; paediatric.
Pain; postoperative.*

Although originally described in 1919 by Kappis [1], paravertebral blockade received little attention until 1979 when Eason and Wyatt [2] reported promising clinical results with a continuous catheter technique. Since then a number of studies of this method in adults have been published [3–6].

Despite the increasing interest in paediatric regional anaesthesia, continuous paravertebral block (CPVB) has received little or no attention [7–12]. Inspired by encouraging results published by Giesecke *et al.* [3] regarding pain relief and diminution of stress response in adult patients treated with CPVB, it was decided to use this technique for a number of selected surgical procedures in children. This paper reports on the initial experience gained in these patients.

Methods

Five children scheduled for renal surgery or cholecystectomy were selected to receive a continuous paravertebral block for intra- and postoperative analgesia. Informed parental consent for inclusion in the study was obtained prior to surgery.

Anaesthetic technique. Intravenous access was secured and anaesthesia induced with thiopentone (5 mg.kg^{-1}). Intubation was facilitated with atracurium (0.5 mg.kg^{-1}) and fentanyl ($1.5\text{--}3.0 \mu\text{g.kg}^{-1}$) and anaesthesia was maintained with isoflurane ($0.5\text{--}1.0\%$), oxygen 33% and nitrous oxide 66% .

Paravertebral block. After intubation the patient was placed in the lateral position with the side to be blocked uppermost. A continuous paravertebral block was performed using a modification of the technique described by Eason and Wyatt [2]. Under aseptic conditions the skin was punctured $1\text{--}2 \text{ cm}$ lateral to the spinous process of T_{7-9} using a paediatric epidural needle (Minipack, Portex: Tuohy needle, 1.08 mm outside diameter; catheter, 0.63 mm outside diameter). The needle was inserted at a 90° angle to the skin in all planes and was advanced until contact was made with the transverse process, then the needle was 'walked' over the top of the transverse process.

The paravertebral space was identified, as the costo-transverse ligament was pierced, by a loss of resistance technique using a saline-filled syringe. Some minor manipulation of the Tuohy needle was usually necessary in order to be able to insert approximately $2\text{--}3 \text{ cm}$ of catheter into the paravertebral space. The catheter was aspirated to check for the presence of cerebrospinal fluid, blood or air and then securely taped to the skin. A test dose of 1.5 ml of bupivacaine 0.25% with adrenaline 1 in $200\,000$ was administered. After approximately 5 min , an initial dose of 0.5 ml.kg^{-1} of the same solution was injected in increments over $5\text{--}10 \text{ min}$. A maintenance dose of 0.25 ml.kg^{-1} was then given hourly for the duration of surgery. At the end of surgery, the position of the catheter was checked by antero-posterior and lateral chest X rays after the injection of 2 ml of radio-opaque dye.

Postoperative management. The patients were observed in the recovery room for an extended period in order to evaluate the effect of the CPVB. The block was maintained by either hourly boluses or by a continuous infusion of approximately $0.25 \text{ ml.kg}^{-1}.\text{h}^{-1}$ of bupivacaine 0.25% with adrenaline 1 in $200\,000$. Morphine $0.05\text{--}0.1 \text{ mg.kg}^{-1}$ was charted to be given intravenously as required at the discretion of the recovery room nurse.

Results

The demographic data of the patients and the details of the blocks are presented in Table 1.

Peroperative course. There were no apparent haemodynamic changes after initiation of the block; similarly no changes were seen at the time of skin and abdominal wall incision. Visceral manipulation led in all patients to an increase in heart rate and blood pressure compared to previous baseline values. However, these changes were easily treated by either an increase in inspired isoflurane concentration ($0.5\text{--}1\%$) or a bolus of fentanyl ($1.5\text{--}3 \mu\text{g.kg}^{-1}$). During closure of the wound, it was possible to reduce the isoflurane concentration to a minimum (0.25%) without any problems. Extubation was smooth and uneventful in all cases.

Table 1. Demographic data and details of continuous paravertebral block (CPVB).

No	Sex	Age; year: month	Weight; kg	Type of surgery	Duration of CPVB after operation; h	Morphine requirement 0–12 h after operation; mg.kg ⁻¹	Spread of dye; no. of ribs
1	M	6:1	20.2	Pyeloplasty	8.5	0.0	4
2	F	1:2	11.0	Pyeloplasty	21	0.1	4
3	M	0:7	9.9	Heminephrectomy	18.5	0.1	6
4	M	3:8	14.4	Cholecystectomy	0	0.4	—
5	M	8:8	26.6	Pyeloplasty	18	0.1	4

Postoperative period. The patients were observed in the recovery room for 8.5–21 h postoperatively. Apart from the immediate postoperative period, the patients were awake and alert. There was no nausea or vomiting.

Patient No. 1 required no additional analgesia during his stay in the recovery room. Patient No. 2 was given one dose of intravenous morphine (0.1 mg.kg⁻¹) 7 h post-operatively and was then comfortable for another 14 h until she returned to the ward. Patient No. 3 was given morphine (0.05 mg.kg⁻¹) at 6.5, 9 and 13 h post-operatively, mainly because of inability to settle. The patient was subsequently given intravenous diazepam (0.5 mg) and went to sleep. In patient No. 4 it was impossible to insert more than a few millimetres of catheter beyond the eye of the Tuohy needle and the catheter was later accidentally dislodged during preparation for the postoperative chest X ray. This patient received the equivalent of 0.6 mg.kg⁻¹ of morphine (morphine 3 mg, pethidine 30 mg) during the first 24 h. Patient No. 5 received one dose of morphine (0.1 mg.kg⁻¹) at 9.5 h post-operatively and needed no further parenteral analgesia for the remainder of the observation period.

Postoperative chest X ray. The standard dose of 2 ml of radio-opaque dye was found to cover four–six adjacent ribs with the largest number being in the smallest patient. The

spread of the contrast media was unilateral and longitudinal in all patients (Fig. 1).

Discussion

Continuous paravertebral block is only suitable for surgery entailing a unilateral incision e.g. cholecystectomy, renal surgery and thoracotomy. Procedures that involve midline incision of the abdominal wall are not appropriate for this technique due to the bilateral innervation of the area. Since CPVB only blocks somatic sensation, pain from visceral structures within the peritoneal cavity and the retroperitoneal space will be left unblocked; a feature displayed by the need to increase slightly the depth of anaesthesia during visceral manipulation. The minor demand for post-operative opioid administration might also be explained by unblocked visceral pain.

Contrary to findings in adult series [4,5], the spread of the radio-opaque dye (and presumably the local anaesthetic) was unilateral, longitudinal and confined to the paravertebral space in the four successful cases; this might imply easier communication between adjacent segments of the paravertebral space in children as compared to adults.

Epidural anaesthesia has been shown to be an excellent block in children [13–15] but has some disadvantages compared to CPVB. If performed at the same level as a CPVB, i.e. in the thoracic region, there is the obvious risk of spinal cord injury. This problem can be circumvented by advancing a lumbar or even a caudal epidural catheter to the thoracic level, as has been advocated by some authors [16–18]. However, this practice has been shown to be relatively unreliable, especially in the smallest children [19]. CPVB, by sparing the lumbar and sacral nerve roots, avoids urinary retention and the uncomfortable and distressing sensation of numb and partially paralysed legs that may occur with an epidural. Although there is a small risk of dural puncture when performing a CPVB [2], this risk is less than that associated with an epidural block. Intravascular injection of local anaesthetic solution is a risk with both techniques and a test dose is recommended. A hazard of performing a CPVB is puncture of the pleura; if this occurs it should be possible to convert the technique into an intrapleural block. Intrapleural block has been shown to be an effective technique in children [20,21] although caution is recommended if planning a prolonged continuous infusion according to the method described by McIlvaine and colleagues [20,21] since this can result in potentially toxic plasma levels of bupivacaine [11]. Technically, CPVB is relatively easy to perform, the only problem being the need to manipulate the Tuohy needle in order to be able to insert 2–3 cm of the catheter beyond the tip of the needle. This could not be achieved in patient No. 4 and the catheter was subsequently accidentally dislodged when moving the patient.

Despite the positive effects of CPVB reported in adult studies [2,3], it has received no attention in recent reviews or textbooks on paediatric regional anaesthesia [7–11]. Without referring to any paediatric studies, Dalens states

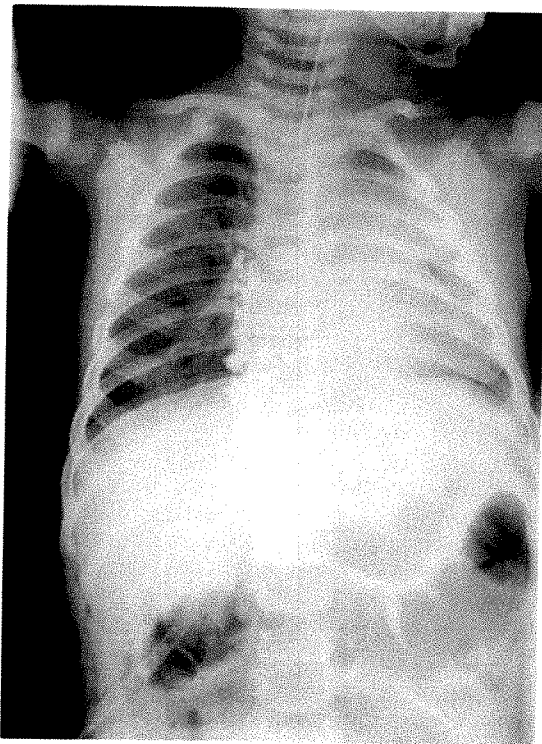


Fig. 1. Autero-posterior chest X ray in a 7-month-old child with a right-sided continuous paravertebral block. The catheter cannot be seen due its very small size.

that 'paravertebral blocks have very few indications in paediatrics' and that 'even in experienced hands, the technique is very hazardous with few benefits for the patient in comparison to other regional procedures especially epidural anaesthesia' [12]. Based on the initial experience with this block in children, the author strongly disagrees with this opinion and believes that CPVB can offer excellent intra- and postoperative analgesia in selected patients, but additional studies are necessary to investigate the technique further and determine its place within paediatric regional anaesthesia.

Acknowledgment

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Factors influencing epidural catheter migration

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Summary

The incidence and amount of migration of epidural catheters was investigated in a prospective randomised study of 153 women who required analgesia in labour. Inward or outward migration occurred in 36% of patients. Inward migration by 1–3 cm occurred in 21 (13.7%) patients and outward migration by 1 cm or more occurred in 34 (22.2%); three (2%) catheters migrated out through the skin. There were significant positive correlations between outward migration and weight, body mass index, and depth of the epidural space. There was no relationship between migration and height, age, intervertebral space used or duration of catheterisation. Problems with epidural block were no more likely in patients in whom migration of 1 cm or more occurred compared with those in whom migration was limited to a maximum of ± 0.5 cm. However, the pattern of problems was different. All cases of failed epidural block occurred in patients whose epidural catheter migrated outward by 2.5 cm or more. Unilateral blockade was not more likely if migration of 1 cm or more occurred.

Key words

Equipment; epidural catheters.

Complications; migration.

Epidural catheters are used during labour in approximately 30% of patients who deliver in our hospital. Migration of the epidural catheter after it has been fixed to the skin has already been shown to occur [1]. Hazards of migration include inadvertent intravenous [2,3] or subarachnoid [4,5] injection and unilateral or failed block [6]. A fixation method with a low incidence of migration, and a means of predicting patients in whom migration is likely, are both desirable. Duffy [7] reported that none of 200 catheters migrated when secured with Opsite transparent dressing. We collected data in order to identify factors which might influence the degree of epidural migration. We used three different standard techniques of fixing the catheter to the skin.

Patients and methods

Women who requested analgesia in normal labour, and those in whom epidural analgesia was indicated for medical reasons (breech presentation, intra-uterine death, or pregnancy-induced hypertension) were eligible for entry into the trial. Patients were not studied if the coagulation profile was abnormal, if it was not technically possible to site the epidural catheter with the patient in the left lateral position, if epidural analgesia was ineffective after injection of 10 ml bupivacaine 0.5% and 10 ml bupivacaine 0.25%, or if dural puncture occurred.

Patients were allocated randomly to receive one of three methods of skin fixation according to a previously prepared list. The age, height and most recent weight in the antenatal clinic were recorded for each patient, and body mass index (BMI) calculated from the equation: $BMI = \text{weight (kg)} / \text{height}^2(\text{m})$.

After establishing an intravenous infusion of compound sodium lactate solution an epidural catheter was inserted by one of four anaesthetists, with the patient in the left lateral position. The L_{2-3} or L_{3-4} interspace was identified by palpation of the iliac crests and spinous processes.

Lignocaine 1% in a volume of up to 10 ml was used to infiltrate the skin and subcutaneous tissue and the skin was incised using a number 11 blade. A 16-gauge Tuohy needle and a catheter from a Portex Minipack, both graduated at 1-cm intervals, were used. The epidural space was identified by loss of resistance to air using a midline approach with the needle bevel facing the right flank. The bevel was then turned cranially and the distance from the skin to the epidural space noted. The epidural catheter was threaded through the needle and the needle removed. The catheter was adjusted to leave 3 cm in the epidural space, and the length of catheter which remained deep to the skin was recorded to the nearest 0.5 cm. A test dose of 3 ml bupivacaine 0.5% plain was given and the catheter was fixed to the skin using one of the three methods described below.

Method 1. The epidural catheter was formed into a circular loop at the skin exit site and then directed over the right shoulder. The skin and catheter were sprayed with 'Opsite' plastic spray (Smith & Nephew Medical Limited). A dressing of two green gauze swabs was placed over the loop and these swabs were sprayed with Opsite and covered with Sleek adhesive tape.

Method 2. The catheter was looped, directed and sprayed as in Method 1. The dressing was transparent 10 × 12 cm Opsite adhesive plastic dressing (Smith & Nephew Medical Limited). No gauze swabs were used.

Method 3. The epidural catheter was looped and directed as in Method 1. A green gauze swab was placed above the skin exit site and another one below, so that the swabs lay between the loop and the skin. A third gauze swab over the loop was used to sandwich the catheter. These swabs were sprayed with Opsite and covered with Sleek adhesive tape.

After taping the catheter over the right shoulder the position of the patient was changed to recline at 45° and a further dose of 7 ml bupivacaine 0.5% plain was given. If the patient had inadequate analgesia at 30 min, the pattern of block was assessed and a further dose of 10 ml bupivacaine 0.25% was given with the patient in an appropriate

Table 1. Distribution of catheter insertion, migration and catheter removal among investigators.

	Doctor A	Doctor B	Doctor C	Doctor D
Number of epidurals	73	58	14	8
Mean (SD) migration; cm	-0.3 (1.0)	+0.7 (2.1)	+0.3 (1.0)	+0.1 (1.0)
Number removed by same doctor	52	51	8	5

position. If this dose was unsuccessful, she was not studied. Further top-up doses of 10 ml bupivacaine 0.25% for first stage analgesia and 10 ml bupivacaine 0.5% for instrumental delivery were given by the attending anaesthetist and their efficacy was recorded. Maternal posture at delivery was recorded.

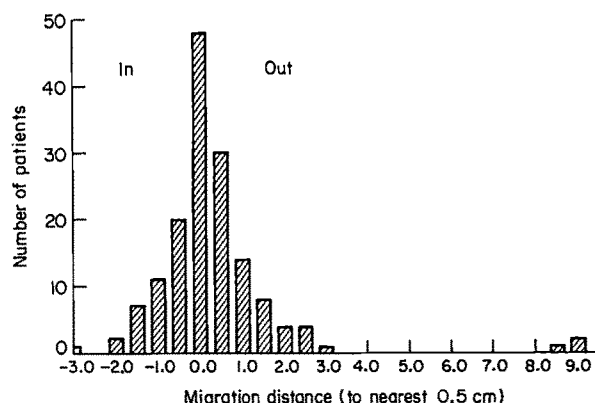
Following delivery and any perineal suturing, the patient was positioned in the left lateral position as for insertion of the catheter and the epidural catheter was removed by the duty obstetric anaesthetist. The length of catheter deep to the skin was recorded to the nearest 0.5 cm. Inward migration was given a negative, and outward migration a positive, value. The results were analysed by one-way analysis of variance or the Chi-squared test to evaluate differences between the different methods of fixation, and by linear regression analysis of data from all patients to evaluate the effect of each factor studied.

Results

One hundred and fifty-three women completed the study. The four authors inserted all epidural catheters (Table 1). One hundred and sixteen (76%) of the catheters were removed by the same anaesthetist who inserted it and another five (3%) were removed by other authors. The remaining 32 (21%) were removed by one of 12 other duty anaesthetists. There were no differences among authors in the degree of migration (ANOVA $p = 0.08$). Fifty-five (36%) of the catheters migrated 1 cm or more from their original position. The mean (SD) migration was +0.3 cm (1.5) with a range -3.0 cm to +9.0 cm (Fig. 1).

Twenty-one (14%) catheters migrated inwards by 1 cm or more. Ninety-eight (64%) catheters remained within 0.5 cm of their original position and 34 (22%) migrated 1 cm or more out of the skin, including three which migrated totally out, although the dressings were intact on inspection. Of these three, two were fixed using method 2 and one by method 3. The three outward migration values of 8.5, 9 and 9 cm were treated as outliers and not included in the analysis of variance or linear regression analysis.

There were significant correlations between the amount of outward migration and body weight, migration and body mass index, and migration and depth of the epidural space from the skin. The correlation coefficients and the regression equations are shown in Table 2. Not only was there a tendency to outward migration in more obese patients with a deep epidural space, but there was also a tendency to inward migration in thinner subjects with a shallow epidural space. There was no significant correlation between migration and duration of epidural catheter inser-

**Fig. 1.** Distribution of length of migration of epidural catheters.

tion or the height or age of the patients. All three catheters that migrated totally out of the skin were inserted at the L₃₋₄ interspace. In the other patients, there was no difference in migration between catheters inserted at the L₂₋₃ or L₃₋₄ interspaces.

There were no significant differences in height, weight, BMI, level of puncture, depth of the epidural space or duration of catheterisation in the three groups of patients allocated to each method of fixation. There were no significant differences in migration among the different methods of fixation (Table 3). The power to detect a difference of 1 cm migration with a standard deviation of 1.5 cm between groups of 50 patients was 0.90.

There were 11 (20%) problems with analgesia in the 55 patients whose epidural catheter migrated 1 cm or more inwards or outwards. There were 13 (13%) problems with analgesia in the 98 patients whose epidural catheter migrated 0.5 cm or less. There was no incident of sub-arachnoid or intravenous injection in either group (Table 4). In the group in which the catheter migrated 1 cm or more, three epidural catheters were resited because analgesia failed (outward migration of 3, 9 and 9 cm). At removal, a further three catheters were found to have migrated 8.5 cm, 3 cm and 2.5 cm outwards and the block had been ineffective for delivery and/or suturing, although the anaesthetist had not been informed. Another four patients required an extra epidural top-up dose for left unilateral blockade; two catheters had migrated in and two migrated out. One catheter was withdrawn to its original position after migrating inwards by 2 cm, associated with right unilateral block.

Table 3. Extent of migration of epidural catheter related to method of catheter fixation.

	Migration			Total
	In	±0.5 cm	Out	
Fixation 1	9	33	7	49
Fixation 2	7	32	13	52
Fixation 3	5	33	14	52
Total	21	98	34	

df = 4; Chi-squared = 3.63; p = not significant

Table 2. Relationships between distance of catheter migration and body mass index (BMI), depth of epidural space from skin and weight of the patient.

Migration = $-3.27 + 0.12$ BMI, correlation coefficient $r = 0.31$, $p < 0.0001$
Migration = $-1.80 + 0.43$ Depth, correlation coefficient $r = 0.22$, $p < 0.005$
Migration = $-2.33 + 0.03$ Weight, correlation coefficient $r = 0.25$, $p < 0.002$

Table 4. Complications of epidural analgesia related to extent of migration of catheter

Problems with analgesia	Migration ≥ 1 cm	Migration ≤ 0.5 cm
	n = 55	n = 98
Extra top-up	4	12
Withdraw catheter	1	0
Failure during/after delivery	3	0
Resite catheter (failure before delivery)	3	0
Caudal blockade	0	1
Total	11	13

In patients whose catheter migrated by 0.5 cm or less, 12 patients required extra epidural top-up doses for unilateral block and one patient required a caudal epidural injection for inadequate perineal analgesia.

There was no significant difference in the number of problems between patients whose catheter migrated 1 cm or more and those in whom the catheter migrated by 0.5 cm or less. There was no significant difference in the incidence of unilateral block between those groups of patients.

Discussion

Previous studies have shown different incidences of epidural catheter migration. Phillips and Macdonald [1] studied 100 patients and found that 46 of the epidural catheters migrated by 1 cm or more. Thirty-one catheters migrated by more than 0.5 cm inwards and 15 catheters migrated by more than 0.5 cm outwards. No failure of epidural analgesia was found to be due to migration out of the epidural space. However, an unstated number of anaesthetists inserted the catheters, there was no standard technique for fixing the catheter, the length of catheter in the epidural space was 2–4 cm, the patients' posture for removal was not stated and the number of midwives who removed the catheters was not stated.

In our study, the incidence of migration of 1 cm or more was 36% (14% inward and 22% outward). The range was 3 cm inward to 9 cm outward. We attempted to reduce between-observer error by using four anaesthetists to site the epidural catheters and standardising the methods of fixation, posture for insertion and posture for removal. However, in only 116 (76%) patients was the catheter removed by the same anaesthetist who inserted it.

Duffy [7] reported 'no untoward movement' of the catheter into or out of the epidural space in 200 obstetric patients and stated that the Opsite transparent adhesive dressing had solved completely the problem of the migrating epidural catheter. One of our three study groups used the same Opsite dressing. The mean (SD) migration was 0.54 (2.0) cm in that group, a value not significantly different from the other two methods of fixation.

We found statistically significant relationships between migration and BMI, migration and body weight and migration and depth to the epidural space. It is possible that a large amount of subcutaneous fat may affect the accuracy of readings of the depth of catheter by allowing more

indentation or tenting of the skin around the catheter exit site, or it may allow greater relative movement between the skin exit site and the point of entry into the supraspinous ligament. This would allow greater migration into or out of the epidural space or allow more catheter to coil up in the subcutaneous tissue. It is not clear why the patients with lower BMI had a tendency to inward migration.

This study showed that the incidence of clinical problems was no different between the patients whose catheter migrated 1 cm or more in or out and the remainder. However, the nature of the problems was different. There were no failures of epidural block in the group whose catheter migrated 0.5 cm or less; epidural block failed in six patients whose catheter migrated outwards by 2.5 cm or more. We suggest that a catheter found to have migrated 2.5 cm or more outwards is unlikely to be effective and should be resited. Fifty-five catheters migrated ± 1 cm or more, and in five of those patients unilateral block occurred. There were 12 cases of unilateral block from 98 catheters which migrated 0.5 cm or less. This suggests that epidural catheter migration is not a major cause of unilateral block. Narang and Linter [8] found that unilateral block was more likely if there was a deep epidural space because of the greater chance of positioning the epidural catheter laterally within the space.

There was only one problem (unilateral block) associated with inward migration of 1 cm or more, which was resolved by withdrawing the catheter by 2 cm, to its original position. We suggest that insertion of more than 3 cm of catheter into the epidural space would reduce the number of failures of analgesia because of outward migration without increasing the incidence of unilateral block. However, the small number of problems associated with inward migration may be related to the catheter becoming coiled in the subcutaneous tissues rather than moving further into the epidural space.

Although securing epidural catheters with a transparent dressing does not reduce the incidence of migration, it does allow inspection of the skin entry site without disturbing the dressing. This would facilitate the detection of catheters which are likely to be ineffective because of outward migration (6 out of 153 in our study).

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Perineuronal morphine in intercostal block

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Summary

In a double-blind, randomised study the potential benefits of combining low-dose morphine with bupivacaine for intercostal nerve blocks for analgesia after biliary surgery were investigated. There was no significant improvement in pain scores or consumption of supplementary analgesics when morphine was added to bupivacaine. This investigation supports the findings of other workers who showed that perineural morphine was ineffective for postoperative pain relief.

Key words

Analgesics, narcotics; morphine.

Anaesthetic techniques, regional; intercostal nerve block.

Since 1981 when Mocavero first reported an analgesic effect of perineurally administered morphine, several case reports and a controlled study have lent support to his observation [1–5]. Although most of these concern patients suffering from chronic pain, one case is reported in which morphine was used in an intercostal 'block' to reduce the pain of multiple rib fractures [6]. In this patient, morphine (5 mg in 10 ml isotonic saline) produced effective analgesia for not less than 48 h. The mechanism is thought to involve the uptake of morphine into the nerve axon followed by centripetal spread to the medullary opioid receptors [1–6].

Other results, however, are contradictory, as two randomised, prospective studies have been reported in which morphine administered as a peripheral 'block', was shown to be ineffective in relieving postoperative pain [7, 8].

Unilateral intercostal block with local anaesthetic is an established method of providing effective postoperative pain relief after subcostal incisions. Apart from the risk of pneumothorax, its only real drawback is its relatively limited duration [9, 10]. The aim of this investigation was to examine the effect of combining a small dose of morphine with a standard dose of bupivacaine for intercostal block. The possible depressant action of such a drug combination on postoperative oxygenation and breathing mechanics were also studied [11].

Methods

The study was approved by the Ethics Committee of the University of Lund, and informed consent was obtained from all patients. The purpose and proper use of the visual analogue scale (VAS) was explained to each patient before operation and all patients were encouraged to ask freely for supplementary postoperative analgesics if needed. A pre-operative chest X ray was performed.

Immediately pre-operatively the patient was instructed in the use of the Wright peak flow meter, and a baseline value of the peak expiratory flow (PEF) was established by selecting the highest out of three measured values obtained with the patient in the semi-sitting position. Following premedication with midazolam (2.5 mg) intravenously, anaesthesia was induced with thiopentone and maintained with nitrous oxide/oxygen and isoflurane. Fentanyl

(0.1–0.4 mg) was given to attenuate the hypertensive response to tracheal intubation and supplementary doses (0.05–0.2 mg) were administered as required. Neuromuscular block was obtained with vecuronium or pancuronium and was reversed by glycopyrronium and neostigmine.

Intercostal block was performed on 50 adult patients, ASA 1 or 2, in the left lateral position before reversal of the neuromuscular block. Moving upwards from the lowest rib palpable with certainty, anaesthetic solution was injected (4 ml) under five separate rib angles. Using a randomised double-blind design, patients received either bupivacaine 0.5% (20 ml) (BP) ($n = 24$), or bupivacaine 0.5% (20 ml) with morphine (4 mg) (BM) ($n = 26$). All blocks were performed by the same anaesthetist.

The patients were kept under observation for 24 h post-operatively with continuous ECG and pulse oximetry monitoring. Peripheral oxygen saturation (SpO_2), pulse and respiratory rates were recorded hourly. PEF values in the semi-sitting position were recorded postoperatively at 1, 4 and 20 h and the highest value obtained from three trials was recorded. A chest X ray was performed after 20 h postoperatively. All patients received oxygen $2 \text{ l} \cdot \text{min}^{-1}$ by nasal prongs for the first 2 postoperative hours. Thereafter oxygen was administered only when needed to keep the SpO_2 over 90%.

The intensity of pain was assessed hourly. The VAS used has a 100 mm, nongraded scale ranging from 'no pain' to 'worst pain imaginable'. On the reverse side cm markings represent the degree of pain as follows: 1–2, slight pain; 3–4, moderate pain; 5–6, rather severe; 7–8, severe and 9–10, unbearable pain (ACO AB, Sweden). When using the scale the patients can adjust a moveable slide to a point which represents the degree of pain; the corresponding values can then be read from the back of the scale. Slide positions between two figures were rounded off to the next higher cm mark. The postoperative ward nurses were encouraged to administer supplementary analgesics liberally by the intravenous or intramuscular route according to their judgement and expertise. The standard analgesic in our institution is 'Ketogan', a combination of ketobemidone (a synthetic opioid) with a spasmolytic. The dose and time of administration were recorded together with details of any antiemetics used (droperidol or dixyrazin).

Table 1. Combined patient and peri-operative data. Values are expressed as mean (SD).

	Bupivacaine	Bupivacaine/ morphine
Male/female	13/11	11/15
Operation; C/CL*	18/6	22/4
Age; years	57 (15.2)	54 (14.7)
Duration of operation; min	128 (40.6)	112 (42.3)
Intra-operative fentanyl; mg	0.3 (0.1)	0.3 (0.1)
Postoperative		
Pulse rate	79 (9.4)	79 (12.3)
Respiratory rate	18 (1.9)	16 (2.3)
Spo ₂ (%)	96 (1.2)	96 (1.5)
Pain free interval; h	10.0 (8.0)	8.2 (7.0)
Ketomebidone; mg:		
1-10 h	3.2 (3.7)	3.6 (3.7)
11-24 h	8.1 (6.4)	10.4 (7.4)
Droperidol; mg	0.7 (2.0)	0.9 (1.5)
Dixyrazin; mg	2.7 (5.2)	2.7 (4.7)
Chest X ray changes (n)	5	6

*C = cholecystectomy, CL = choledocholithotomy.

All measurements and recordings in the postoperative period were performed by trained intensive care nurses.

A group of patients was sought which conformed to three criteria indicating a high-quality block, i.e. no supplementary analgesics required over the first 5 postoperative hours, no VAS score over 6 cm on any occasion during this period and no cumulative total score exceeding 15. The groups and subgroups were compared separately using the unpaired Student's *t*-test.

Results

Patient data and peri-operative conditions for the total population are shown in Table 1. There were no significant differences in any of the observations (pulse rate, respiratory frequency, Spo₂). The mean (SD) pain-free interval (time from block to first supplementary analgesic with no VAS score > 6) was 10.0 (8.0) h in the plain bupivacaine group and 8.2 (7.0) h in the bupivacaine plus morphine group. There were no differences in mean VAS scores or

Table 2. Combined patient and peri-operative data in both subgroups with effective blocks for the first 5 postoperative hours. Values are expressed as mean (SD).

	Bupivacaine	Bupivacaine/ morphine
Male/female	8/6	7/5
Operation; C/CL*	12/2	10/2
Age; years	59 (13.0)	53 (16.5)
Duration of operation; min	123 (30.9)	113 (32.8)
Intra-operative fentanyl; mg	0.3 (0.1)	0.2 (0.1)
Postoperative		
Pain free interval; h	15.1 (7.2)	13.8 (5.7)
VAS scores		
1-10 h	1.1 (1.1)	1.3 (0.8)
11-24 h	2.1 (1.6)	2.4 (1.4)
Ketomebidone; mg		
1-10 h	1.5 (2.5)	0.8 (1.6)
11-24 h	6.6 (6.5)	10.1 (9.6)
Chest X ray changes (n)	2	3

*C = cholecystectomy, CL = choledocholithotomy.

mean consumption of analgesics and antiemetics. Five patients in the bupivacaine and six in the bupivacaine plus morphine groups showed slight radiographic abnormalities (minor atelectasis or pleural effusions). No pneumothorax was seen and no signs of central opioid side effects such as itching or urine retention were observed.

Twenty-six patients conformed to the criteria for a high quality block. Fourteen of these had received bupivacaine alone, compared to 12 in the combined solution group (Table 2). There were no significant differences in any observations between the groups. Mean VAS scores and mean consumption of supplementary ketobemidone during the early and late phases of the observation period are also given in Table 2. The number of patients still remaining pain free for each postoperative hour (i.e. those with VAS scores < 6 and no supplementary analgesics) is shown in Figure 1. The changes in respiratory mechanics, when measured as a percentage fall in PEF for the patients in the two groups who were able to cooperate adequately, were of about the same order of magnitude (Table 3).

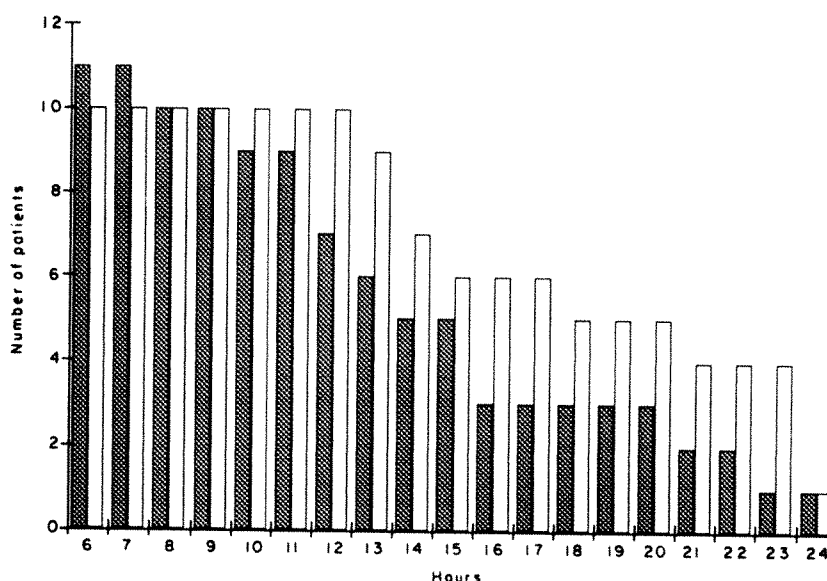
**Fig. 1.** Number of patients remaining pain free (i.e. no need for supplementary analgesia and no VAS pain score > 6) at each hour between 6 and 24 h postoperatively. □, plain bupivacaine; ▨, bupivacaine plus morphine.

Table 3. Mean (SD) percentage falls in PEF from pre-operative values in the two groups at the times shown postoperatively.

	1 h	4 h	20 h
Bupivacaine (n = 7)	47 (18.4)	39 (18.7)	47 (13.0)
Bupivacaine/morphine (n = 11)	44 (13.7)	42 (9.8)	49 (14.6)

Discussion

Postoperative analgesia with epidural or spinal blocks using local anaesthetics and/or opioids are both well-proven, invasive procedures, with a small, but real, risk of serious complications. The idea of providing comparable duration of analgesia by means of a series of relatively simple nerve blocks motivated this study.

It is well known from autopsies and radiographic studies that a solution correctly deposited as an intercostal block spreads extensively behind the posterior intercostal membrane, and that diffusion into the epidural space may occur [10, 12, 13].

A crucial point in an investigation of this kind is to ensure that the morphine solution has been correctly deposited in the immediate vicinity of the nerve. By mixing the morphine and bupivacaine and by comparing only the initially successful blocks, we are satisfied that the intercostal nerves had been adequately exposed to the morphine component. Also, the initial local anaesthetic effect would cover the possibly slower onset of the opioid. There is no evidence that bupivacaine might hinder the axonal uptake of morphine as these drugs show mutually potentiating effects [14, 15].

The mean duration of a conventional intercostal block with bupivacaine could be expected to be about 10–15 h [9, 10]. The clinical evaluation of postoperative pain is certainly not an easy undertaking, and in our study the observations were made by several different observers. However, we still believe that taken together the VAS scores and the consumption of supplementary analgesics would have revealed some differences, at least in the later phase of the observation period, if any clinically useful analgesic effect of the morphine component had been present.

We must conclude that low-dose morphine (4 mg) with bupivacaine 0.5% for intercostal nerve block is not a clinically useful method of prolonging postoperative pain relief.

Acknowledgments

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Haemodynamic stability during general anaesthesia for intra-ocular surgery: the effect of topical oxybuprocaine

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Summary

Local anaesthesia is frequently used in combination with light general anaesthesia to reduce the reflex responses to surgical stimulation. This combination has not previously been evaluated for intra-ocular surgery. During cataract extraction under general anaesthesia, the effect of topical anaesthesia with oxybuprocaine 0.4% on the pressor response was compared with normal saline in a control group. The simple technique of instilling local anaesthetic drops into the conjunctival sac blocked the pain pathway sufficiently to prevent the pressor response to surgical stimulation ($p < 0.001$). Higher inspired concentrations of enflurane were required in the control group to achieve and maintain haemodynamic stability ($p < 0.001$).

Key words

Anaesthetics, local; oxybuprocaine.
Surgery; ophthalmological.

Although the use of local anaesthesia for cataract surgery is increasing [1, 2] there are many patients for whom general anaesthesia will be preferred. Surgeons require both an immobile, uncongested operative field and a constant low intra-ocular pressure. Increases in systolic arterial pressure in response to surgical stimulation may increase bleeding [3] and intra-ocular pressure [4–6]. Under general anaesthesia this pressor response is normally controlled by the use of volatile anaesthetic agents and opioid analgesics. This study investigates whether topical anaesthesia of the eye combined with light general anaesthesia blocks the pain pathway sufficiently to prevent an increase in arterial pressure when the surgical stimulus is applied.

Methods

Thirty-seven patients of ASA grade 1 or 2 scheduled for cataract extraction and intra-ocular lens implantation were randomly allocated to one of two groups. All patients gave informed consent and the study was approved by the local ethics committee.

The patients were premedicated with temazepam 10 mg given 1 h before operation. Anaesthesia was induced with fentanyl $1 \mu\text{g.kg}^{-1}$ and thiopentone in a dose sufficient to abolish the eyelash reflex. Vecuronium 0.1 mg.kg^{-1} was given to facilitate orotracheal intubation and maintain neuromuscular blockade. Anaesthesia was maintained with enflurane in 70% nitrous oxide in oxygen. Ventilation was controlled to maintain normocapnia ($P_{\text{ET}}\text{CO}_2$ 4.8–5.4 kPa) as indicated by a Datex Capnograph.

During induction, patients in group 1 were given three drops of oxybuprocaine 0.4% and those in group 2 three drops of sodium chloride solution 0.9% into the conjunctival sac of the eye for operation. The identity of the drops was concealed from the investigators.

Following induction of anaesthesia, serial systolic arterial blood pressure measurements were taken using an oscillotonometer [7]: before surgery (t1), during insertion of the eyelid retractor (t2), during insertion of the superior rectus muscle stay suture (t3), during corneal incision (t4) and at 2, 3, 4, 5 and 10 min thereafter (t5–9). Enflurane was administered from a calibrated mark 3 enflurane vaporizer (Ohmeda), at a concentration setting required to achieve a systolic arterial pressure as close as possible to the pressure at t1 (before surgery).

The mean and standard error of the systolic arterial blood pressure and enflurane concentrations of all the patients in each group at each of the times t1–9 were calculated. Student's unpaired *t*-test was used to assess the difference in systolic arterial pressure and enflurane concentration between the two groups at each time point. Statistical significance was defined as $p < 0.001$.

Results

Twenty patients received oxybuprocaine drops and 17 patients 0.9% sodium chloride solution. The groups were similar in respect of age, weight and sex (Table 1). There was no significant difference in the mean systolic arterial blood pressure of the two groups at t1.

In the oxybuprocaine group there was no significant change in the mean systolic arterial pressure during surgery (t2–t9) compared with the pressure before surgery (t1). In the saline group, however, the mean systolic arterial pressure increased when surgery commenced and did not return to the presurgery level until 10 min had elapsed. These differences were highly significant from t4–t6 ($p < 0.001$) (Fig. 1).

The saline group required higher settings of the enflurane vaporizer to control the increase in systolic arterial pressure and this was significant from t4 when the corneal incision was made until t6 3 min after surgery commenced (Fig. 2).

Discussion

This study demonstrates that the application of local anaesthetic eye drops to the conjunctival sac blocks the pain pathway sufficiently to prevent an increase in arterial pressure during cataract surgery under light general anaesthesia without the need to increase inspired enflurane concentration. Increasing the depth of anaesthesia in

Table 1. Patient characteristics, mean (SEM or range).

	Oxybuprocaine	Saline
Age; years	71.8 (85–51)	74.4 (85–55)
Weight; kg	70.0 (2.57)	74.2 (3.15)
Males/females	7/13	5/12

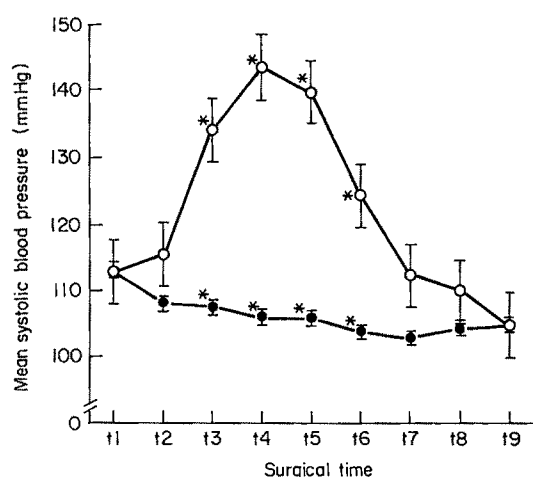


Fig. 1. Systolic arterial pressure, mean (SEM) during initial 10 min of surgery. ● = group 1 oxybuprocaine, ○ = group 2 saline. *significant difference between groups ($p < 0.001$).

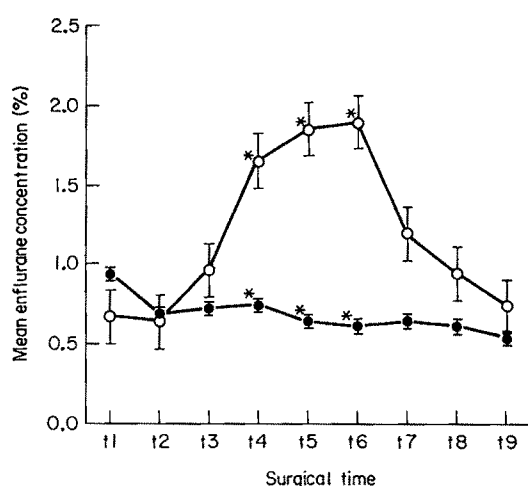


Fig. 2. % enflurane concentration, mean (SEM) during initial 10 min of surgery. ● = group 1 oxybuprocaine, ○ = group 2 saline. *significant difference between groups ($p < 0.001$).

anticipation of a painful stimulus is likely to cause unwanted hypotension during the period before surgery commences, particularly in the elderly [8]. Ageing increases the haemodynamic response of incision [8] therefore the elderly population requiring cataract extraction are likely to obtain increased benefit from a technique which minimises afferent neuronal impulses from the operation site.

Local anaesthetic techniques are commonly used either alone [9], or with general anaesthesia in many branches of surgery to obtund reflex responses to surgical stimulation, particularly in the high risk patient [10]. The use of local anaesthetic techniques to supplement general anaesthesia has not previously been described for intra-ocular surgery. Retrobulbar [11] and peri-orbital block [2] are invasive procedures, requiring expertise and practice and are not without risk. They normally would not be contemplated without the added benefits of avoiding a general anaesthetic. Topical anaesthesia is simple and noninvasive and blocks the rich supply of sensory nerve endings in the cornea and conjunctiva. It is normally used for surface procedures such as tonometry, removal of sutures and superficial foreign bodies and on its own is not adequate for intra-ocular surgery. The addition of light general anaesthesia and neuromuscular blockade renders the eye

immobile and provides suitable conditions for cataract surgery. Oxybuprocaine is a benzoic acid ester related to procaine and is in common use in ophthalmology [12]. Anaesthesia of the cornea develops within 5 min and sensitivity returns to normal after approximately 1 h.

In this study we wanted to show that haemodynamic stability was achieved without the need for continual adjustment of the inspired concentration of enflurane. We therefore chose to record the vaporizer setting as an indicator of the action that the anaesthetist was taking to achieve haemodynamic stability.

Although low concentrations of enflurane were used in this study, particularly in group 1, there was no incidence of awareness. Isoflurane in air produces loss of recall of neutral words at levels as low as 0.2 MAC in volunteers although 'shock' words required up to 0.4 MAC [13]. The MAC of inhalational agents decreases with age [14, 15] and is further reduced in combination with opioids, nitrous oxide and hypnotics [15]. We studied the first 10 min of surgery because the intensity of the stimulus changes rapidly from zero before surgery starts to a maximum when the superior rectus stay suture is inserted and to a reduced level during intra-ocular manipulation. This change in the intensity of the painful stimulus over a short time interval provides an ideal opportunity to test the efficacy of the local anaesthetic.

In conclusion, topical anaesthesia with oxybuprocaine is a safe and simple method of improving haemodynamic stability during cataract surgery under light general anaesthesia.

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Intravenous regional anaesthesia with 0.5% prilocaine or 0.5% chloroprocaine

A double-blind comparison in volunteers

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Summary

Ten healthy male volunteers received intravenous regional anaesthesia of the upper limb on two separate occasions using 40 ml of 0.5% prilocaine or 40 ml plain 0.5% chloroprocaine by random allocation. Using a double-blind method, the onset and recovery of sensory block was tested (pinprick) in the four main nerve areas. The onset and recovery of motor block was measured by squeezing a rubber balloon connected to a manometer. The symptoms after deflation of the tourniquet were recorded. Seven volunteers in both groups developed total anaesthesia in 20 min. Complete motor block occurred in 12.3 min with prilocaine and 11.3 min with chloroprocaine. Sensation recovered in all areas in 7.6 min with prilocaine and 10.0 min with chloroprocaine (ns). Motor function recovered in 8.4 and 12.0 min respectively ($p < 0.01$). Six volunteers in the chloroprocaine group showed signs of venous irritation and/or antecubital urticaria in the test arm for 30–45 min after the deflation. Four volunteers in the chloroprocaine group had increased in heart rate ($> 20\%$) and one of them short periods of junctional rhythm during the first 2 min after tourniquet deflation. Mild, short-lived CNS side effects occurred in both groups.

Key words

Anaesthetics, local; prilocaine, chloroprocaine.
Anaesthetic techniques, regional; intravenous.

Chloroprocaine has a fast onset of action and is rapidly broken down by plasma cholinesterase. These properties, together with low toxicity, suggest that it might be suitable for intravenous regional anaesthesia (IVRA). In the 1960s chloroprocaine was used for this purpose, but after reports of postanaesthetic thrombophlebitis its use in IVRA ceased [1, 2]. The venous irritation may have been caused by the preservative (methylparaben) and/or anti-oxidant (sodium bisulfite) present in the local anaesthetic solution. The low pH (3.1) of the solution may also have contributed to the problem.

Recently, a new 0.5% solution without additives (pH 3.7), has been introduced for IVRA and used successfully in several hundreds of patients with no signs of thrombophlebitis or other serious side effects [3, 4]. This IVRA study in volunteers was designed to compare the new, plain 0.5% chloroprocaine solution with 0.5% prilocaine, which for many years has been our preferred local anaesthetic for IVRA.

Methods

With the approval of the Ethics Committee of the hospital, 10 healthy male volunteers, who gave their informed consent, were studied. They received IVRA of the upper limb on two occasions, separated by an interval of at least 10 days, using 0.5% prilocaine (pH 6.7) (Citanest, Astra, Sweden) 40 ml or plain 0.5% chloroprocaine (pH 3.7) (Sintetica, Switzerland) 40 ml in a randomised double-blind fashion. The intravenous block was performed in a standard manner using a double-cuffed tourniquet. Analgesia and anaesthesia were assessed at four separate innervation areas corresponding to particular sensory nerves using a pinprick technique before the block and at 2, 5, 7.5, 10, 15 and 20 min after injection of the local anaesthetic.

Analgesia was considered to be present when a sharp needle applied to the skin was experienced as blunt touch. Anaesthesia was said to have occurred when there was no sensation. Motor blockade was evaluated by hand-squeezing with maximal force a rubber balloon connected to a manometer (Martin Vigorimeter, Germany) at the same testing intervals. Complete motor block was recorded when the volunteer could not induce any movement on the manometer gauge. Twenty minutes after injection of the test solution, the tourniquet was deflated in one step and analgesia and motor blockade were subsequently evaluated at 2, 4, 6, 8, 10, 15 and 20 min after deflation. Any CNS or other side effects were noted. The ECG was monitored continuously and recorded on paper just before cuff deflation and for 2 min thereafter.

Statistical analysis between the mean values was performed using paired Student's *t*-tests and frequencies were compared by Fisher's Exact test. A *p* value of < 0.05 was considered statistically significant.

Results

With both drugs, seven volunteers showed total anaesthesia at all the tested sites within 20 min. There were only small differences in the development of analgesia in the different nerve areas between the groups (Tables 1 and 2). All the volunteers developed complete motor block on each occasion, after 12.3 (SD 3.6) (range 7.5–15) min with prilocaine and 11.3 (4.3) (5–15) min with chloroprocaine (ns). After deflation of the tourniquet, sensation recovered in all nerve areas in 7.6 (4.9) (4–20) min with prilocaine and in 10.0 (2.9) (6–15) min with chloroprocaine (ns). Motor function recovered to pre-anaesthetic values in 8.4 (4.3) (6–20) min with prilocaine and in 12.0 (4.6) (6–20) min with chloroprocaine ($p < 0.01$).

Table 1. Mean (SD) (range) times (min) to analgesia of the nerves shown.

	Ulnar	Radial	Median	Musculo-cutaneous
Chloroprocaine	9.9 (7.3) (2-20)	7.5 (4.5) (2-15)	8.2 (5.7) (2-20)	4.6 (2.4*) (2-10)
Prilocaine	11.0 (6.8) (2-20)	6.5 (1.8) (5-15)	9.8 (4.6) (5-15)	7.5 (4.6*) (5-20)

* $p < 0.05$ between the groups.

Table 2. Mean (SD) (range) times (min) to sensory recovery of the nerves shown.

	Ulnar	Radial	Median	Musculo-cutaneous
Chloroprocaine	6.5 (4.2) (2-15)	6.4 (2.5) (4-10)	5.8 (2.7) (2-10)	8.7 (2.9) (4-15)
Prilocaine	4.2 (1.1) (2-6)	4.4 (2.3) (2-8)	4.2 (1.8) (2-8)	7.2 (5.3) (2-20)

Local anaesthetic toxicity reactions were more frequent with chloroprocaine. In six of the volunteers, after the chloroprocaine block, signs of venous irritation (distention, purple coloring, itching) and/or urticaria in the antecubital skin area of the exposed arm occurred for about 30-45 min. There were no similar side effects after prilocaine block ($p < 0.01$). During the next 10 days, no signs of thrombophlebitis were observed. Mild CNS side effects (light-headedness, dizziness) occurred from approximately 45 s to 5 min after tourniquet cuff deflation on two occasions after prilocaine and five times after chloroprocaine (ns). One volunteer, when receiving chloroprocaine, had several short periods of junctional rhythm on the ECG during the first 2 min after deflation of the tourniquet; four volunteers experienced an increase in heart rate (20% of pre-operative value) when chloroprocaine was used, compared with none after prilocaine IVRA ($p < 0.05$).

Discussion

There was no clinically significant difference in the efficacy of anaesthesia and recovery from anaesthesia between 0.5% prilocaine and plain 0.5% chloroprocaine in the doses stated when used for IVRA (Bier's block). The development of sensory block was similar to that observed earlier in patients who received a similar dose of prilocaine [5]. Venous irritation and urticaria were more frequent after chloroprocaine than after prilocaine, which contrasts with the reports of no side effects when chloroprocaine was used for IVRA in patients [3, 4]. The signs of

venous irritation were short-lived, disappearing within 45 min. Thereafter, there were no inflammatory changes in the veins and no clinical signs or symptoms of thrombus formation was observed.

The urticarial reactions in these volunteers occurred only in the antecubital area; there were no signs of general anaphylactic reactions or of urticaria in other areas. The low pH of chloroprocaine may have a direct toxic effect on the venous endothelium, and when the circulation is restored after tourniquet deflation this may activate mast cells and cause release of vasoactive substances in that area.

Gerber and colleagues reported transient increases in heart rate and instances of junctional rhythm or ventricular ectopic beats after chloroprocaine IVRA of the leg [4]. Whether this is the result of a hyperaemic flush of acidic blood from the arm into the circulation or a direct cardiac effect of chloroprocaine remains unknown. The CNS effects were mild after either local anaesthetic and their incidence was comparable to those observed in our previous studies with prilocaine [5, 6].

In conclusion, in the doses used, 0.5% chloroprocaine produces IVRA as effectively as 0.5% prilocaine. Chloroprocaine does not appear to have any clinical advantages over prilocaine for IVRA and the venous irritation observed may limit its clinical usefulness. It is disappointing that this new preservative and antioxidant free chloroprocaine solution appears to retain the irritant effect on veins which was such a feature of the old preparation.

Acknowledgments

We thank Mr D. Menon (Sintetica AS) for the supply of 0.5% chloroprocaine. The study was supported by Sigrid Juselius Foundation (P.H.R.).

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The effect of pre-induction glycopyrronium on the haemodynamic response of elderly patients to anaesthesia with propofol

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Summary

This study investigated whether pretreatment with glycopyrronium can attenuate the hypotension caused by anaesthesia of the elderly with propofol. Twenty elderly patients (77.1 ± 2.44 years, mean $\pm$ SEM) of ASA physical status 2 or 3 scheduled for elective urological procedures were given glycopyrronium 0 ($n = 10$) or $5 \mu\text{g.kg}^{-1}$ ($n = 10$) in a randomised, double-blind manner, 5 min before induction of anaesthesia with propofol infused at 600 ml.h^{-1} (average induction dose $1.7 \pm 0.06 \text{ mg.kg}^{-1}$, mean $\pm$ SEM) followed by maintenance with a propofol infusion at $10 \text{ mg.kg}^{-1}.\text{h}^{-1}$. Although glycopyrronium significantly increased heart rate ($p < 0.01$, ANOVA), the decrease in blood pressure 2 and 5 min after induction was similar in both groups. The study had a power of 80% to detect a 20 mmHg difference in systolic arterial pressure between treatment groups with $p < 0.05$.

Key words

Anaesthetics, intravenous; propofol.
Parasympathetic nervous system, glycopyrronium.
Complications; hypotension.

Propofol anaesthesia often causes hypotension and this may be particularly severe in the elderly [1]. When glycopyrronium $5 \mu\text{g.kg}^{-1}$, atropine $10 \mu\text{g.kg}^{-1}$ and placebo were compared as pretreatments given 5 min before total intravenous anaesthesia with propofol and alfentanil, the two anticholinergic agents caused equivalent increases in heart rate but those patients who received glycopyrronium suffered less hypotension than those receiving atropine or saline [2]. This apparent antihypotensive effect was attributed to differences in affinity of atropine and glycopyrronium for the M_1 and M_2 muscarinic receptor subtypes. Subsequently, a larger study compared placebo, 2.5, 5 and $7.5 \mu\text{g.kg}^{-1}$ of glycopyrronium as pretreatments before anaesthesia with propofol and a small dose of fentanyl [3]. The average ages of the patients in these two studies were 42.5 years [2] and 39 years [3] respectively.

We have compared the effects of glycopyrronium $5 \mu\text{g.kg}^{-1}$ and placebo on the haemodynamic consequences of induction and early maintenance anaesthesia with propofol in elderly patients.

Methods

The study was approved by the South Manchester District Ethics Committee and all patients gave informed consent in writing. Twenty patients of ASA physical status 2–3 weighing 35–80 kg and aged 65–85 years who were scheduled for urological procedures (usually check cystoscopy) were recruited into the study. Day surgery patients received no premedication; patients expected to stay overnight received oral temazepam 10–20 mg 100 min pre-operatively.

An anaesthetist who was not otherwise involved in the study mixed glycopyrronium injection and 0.9% saline solution to give a solution containing glycopyrronium 0 or $400 \mu\text{g}$ in 8 ml, and 0.1 ml.kg^{-1} of this solution was administered intravenously in a randomised, double-blind manner 5 min before the start of induction giving a glyco-

pyrronium dose of 0 (group 1) or $5 \mu\text{g.kg}^{-1}$ (group 2). Five min later, anaesthesia was induced by infusing propofol at 600 ml.h^{-1} using an Ohmeda 9000 electronic syringe pump in 'bolus' mode whilst the patient counted aloud (a small pilot study showed that induction at 1200 ml.h^{-1} causes unacceptable hypotension in these elderly patients). Use of the syringe pump allowed a standardised induction for all patients and facilitated the transition to infusion maintenance. Induction of anaesthesia was considered to be completed at the moment of cessation of counting and the pump speed was then reduced to deliver propofol at $10 \text{ mg.kg}^{-1}.\text{h}^{-1}$. After induction, patients breathed 100% oxygen through a Mapleson A anaesthetic breathing system and a face mask. Apnoea lasting more than 30 s was treated by gentle manual inflation of the lungs. Six min after induction the study was terminated and anaesthesia continued with agents and techniques appropriate to the scheduled surgical procedure. No analgesia or intravenous fluids were given before or during the study period. Arterial blood pressure was measured at 6, 4 and 1 min before and 2 and 5 min after the start of induction using an automatic

Table 1. Patient data and induction dose for the two treatment groups.

	Group 1 (control)	Group 2
Glycopyrronium dose; $\mu\text{g.kg}^{-1}$	0	5
Number of patients	10	10
Age; years	76.3 (1.8)	77.9 (2)
Sex	6M/4F	8M/2F
Weight; kg	66.6 (3.7)	59.6 (3.7)
ASA status; 1, 2, 3	(0/8/2)	(0/8/2)
Induction dose of propofol; mg.kg^{-1}	1.65 (0.07)	1.8 (0.01)

There were no significant differences between the groups. Data are means (SEM).

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Table 2. Heart rates, and systolic, mean, and diastolic blood pressures of groups 1 and 2 after propofol induction and early maintenance.*

	Min after induction	Group 1 (control)	Group 2
Glycopyrronium dose; $\mu\text{g.kg}^{-1}$		0	5
Number of patients		10	10
Heart rate; beat.min^{-1}	-6	75.1 (3.90)	71 (3.41)
	-4	76 (3.29)	77.1 (4.02)
	-1	73.8 (4.55)	†81.7 (4.42)
	2	72.7 (3.44)	†84.1 (5.26)
	5	71.3 (2.44)	†80.2 (4.15)
Systolic arterial pressure; mmHg	-6	157.4 (7.83)	154.6 (8.53)
	-4	157.7 (6.61)	153.4 (6.93)
	-1	155.6 (7.58)	146 (8.84)
	2	†115.1 (6.59)	†115.3 (5.76)
	5	†122.3 (7.84)	†116.4 (6.52)
Mean arterial pressure; mmHg	-6	118.1 (4.78)	111.6 (5.84)
	-4	120.5 (5.60)	116.2 (5.49)
	-1	115.8 (5.64)	114.4 (6.85)
	2	†88.2 (6.12)	†83.1 (5.37)
	5	†90.6 (6.58)	†88.3 (6.50)
Diastolic arterial pressure; mmHg	-6	87.9 (4.21)	86.3 (5.42)
	-4	85.9 (3.78)	84.5 (4.79)
	-1	82.9 (4.07)	82.5 (4.40)
	2	†64.6 (4.36)	†66 (3.51)
	5	†69 (4.75)	†70.3 (5.34)

*Glycopyrronium or placebo was given at -5 min and propofol commenced at 0 min. There were no significant differences between the groups. Data are means (SEM).

†Significant change from baseline (-6 min) value $p < 0.05$ ANOVA.

‡Significant change from baseline (-6 min) value $p < 0.01$ ANOVA.

sphygmomanometer (Dinamap) calibrated in accordance with the manufacturer's instructions. ECG (lead CM5) was monitored throughout the study.

Patients' age, sex, weight and ASA status were compared using the Mann-Whitney *U* test. Analysis of variance (Scheffe *F*-test) was used to evaluate within-group and between-group differences in heart rate and blood pressure.

Results

There were no differences in age, weight and induction dose of propofol between the two groups (Table 1). Heart rate and blood pressure were similar in the two groups of patients before administration of glycopyrronium or saline ($p > 0.05$). Heart rate was increased by glycopyrronium ($p < 0.01$) and remained higher than its baseline rate after induction, whereas those patients who received no glycopyrronium showed a slight reduction in heart rate after induction (Table 2).

After induction, systolic, mean and diastolic arterial blood pressures decreased significantly ($p < 0.01$) in both groups, with patients in the glycopyrronium group showing a decrease similar to that in the saline (control) group (Table 2). There were no significant between-group differences in arterial blood pressure at any time during the study. No arrhythmias or ST segment changes were detected in any patient.

Discussion

Although propofol caused a substantial reduction in blood pressure within 2 min of induction, we were unable to demonstrate any haemodynamic benefit from pretreatment with glycopyrronium in our study population.

Our study had a power of 80% to detect a 20 mmHg difference in systolic arterial pressure between treatment groups with $p < 0.05$, and we conclude that if glycopyrronium does have any effect on the hypotension seen at the start of propofol anaesthesia it is too small to be useful.

Pretreatment with glycopyrronium before total intravenous anaesthesia with propofol has now been evaluated in three double-blind controlled studies and a total of 149 patients [2, 3]. Although the original (smaller) study of Skues *et al.* [2] suggested that this technique might be valuable, this has not been borne out by closer investigation and the only valid reason for anticholinergic pretreatment would now appear to be as prophylaxis against bradycardia in selected patients. Reducing the rate of administration of propofol during induction decreases the dose required and minimises the haemodynamic disturbance [4] without compromising the quality of induction. If the hypotensive effect of propofol is primarily due to increased venous capacitance [5], then other rational antihypotensive strategies might include fluid preloading or the use of vasopressors. These have not yet been adequately evaluated.

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Predicting difficult intubation. What factors influence the thyromental distance?

We were interested to read Dr Frerk's article (*Anaesthesia* 1991; **46**: 1005–8) on predicting difficult intubation. Use of the thyromental distance to predict difficult intubation should be taken a step further by assessing what particular anatomical factors have contributed to a shortened distance. Study of the anatomy will reveal three possibilities (Fig. 1). (1) *Head extension*. Extension of the head with the neck flexed brings the axis of the mouth into alignment with the axes of the pharynx and larynx [1]. Inability to extend the head is an important cause of difficult intubation. Thyromental distance depends on head extension. (2) *The length and depth of the mandible*. A receding mandible has been identified with difficult intubation. Deepening of the alveolar–mental distance (anterior depth of the mandible) has also been sited as a cause of difficult intubation, requiring wider opening of the mandible to visualise the larynx [2]. A short mandible or deepening of the alveolar–mental distance will result in a reduced thyromental distance. (3) *The position of the larynx*. A higher anterior larynx is associated with difficult intubation. It is usually only with hindsight that we identify this as a cause of difficult intubation. However, can we predict it pre-operatively? As the vocal cords are attached anteriorly to the thyroid cartilage, a high anterior thyroid prominence may indicate a high anterior larynx. A high anterior thyroid prominence will also result in a shortened thyromental distance.

Appreciating that a patient is going to be difficult to intubate is only part of the problem: we need to ascertain the anatomical reason. If the thyromental distance is to be used to its full value to predict difficult intubation, we should find out which of the above factors has contributed to a shortening of this distance.

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A reply

Thank you for giving me the opportunity to reply to the letter of Dr King and Professor Adams. They have explained clearly and concisely why thyromental distance is such a good test for predicting difficulty, since this one simple measure encompasses limited head extension,

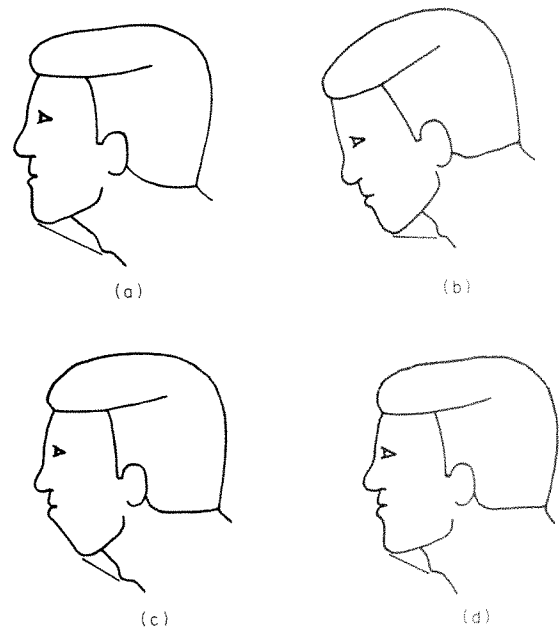


Fig. 1. Factors influencing the thyromental distance. (a) Normal; neck flexed, head extended. (b) Poor head extension. (c) Receding mandible and/or deep alveolar–mental distance. (d) High anterior thyroid cartilage prominence (high anterior larynx).

receding jaw and high anterior larynx. I still believe that as a screening test it should be combined with a Mallampati type test to reduce false alarms, a view supported by a recent editorial [1].

They have suggested that predicting difficulty is only part of the problem and that the anatomical reason for difficulty needs to be determined. While this is true for the anaesthetist who plans to tackle the intubation him or herself, in practice a screening test can be used by many anaesthetists simply to decide that they need to summon experienced help to assist with the intubation, and that is really all they need to decide to ensure patient safety. Further examination at this stage simply complicates an otherwise simple test. It is the experienced helper, who has been called to assist, who needs to assess more accurately the precise nature of the problem in order to ascertain the best way to deal with it.

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The variable deadspace is not necessary

I cannot understand the rationale for the variable deadspace described by Drs Scott and Jones (*Anaesthesia* 1991; **46**: 1047–9). I share their analysis of the goals of controlled ventilation, i.e. firstly to maintain oxygenation, by maintaining an adequate FRC, and secondly to maintain normocapnia or slight hypocapnia, and the authors are clearly not afraid of routinely using PEEP (10 cmH₂O was used in their study) so that mechanical overventilation in order to prevent atelectasis is unnecessary. If a capnograph is agreed to be essential then tidal volume and respiratory

rate can be adjusted to produce a desired level of end-tidal PCO_2 – $PE'CO_2$. Why then ventilate according to body weight? Nomograms are based on two flawed assumptions, that CO_2 production ($\dot{V}CO_2$) and the physiological deadspace fraction are 'normal'. $\dot{V}CO_2$ varies with age, body temperature and depth of anaesthesia. Dead-space also varies considerably. When capnography is available it is better to address the real problem: how to estimate arterial PCO_2 from end-tidal PCO_2 ?

$PaCO_2$ – $PE'CO_2$ increases with age in smokers much more

markedly than in non- and exsmokers [1], and is dependent on tidal volume [2]. I ventilated the lungs of 58 patients at two settings (firstly at 17 breath.min⁻¹, volume 0.4–0.51, and then at 9 breath.min⁻¹, volume 0.7–0.81), so that group mean P_{aCO_2} was the same at both settings. At small volumes, median $P_{aCO_2}-P_{E'CO_2}$ and the 5th and 95th percentiles were 0.6, 0.2 and 1.4 kPa; at large they were 0.3–0.1 and 1.1 kPa. Mean $P_{aCO_2}-P_{E'CO_2}$ was thus 0.3 kPa less at the larger tidal volume; in some young patients $P_{aCO_2}-P_{E'CO_2}$ was small and did not change much with ventilator setting. $P_{aCO_2}-P_{E'CO_2}$ was greater in patients with delayed pulmonary emptying, i.e. airways obstruction.

The capnograph and pulse oximeter allow excellent monitoring of ventilation. Ventilator systems are bulky enough; the variable deadspace is unnecessary.

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A reply

Dr Fletcher is right. Variable deadspace is unnecessary. We never said it was necessary; rather, that given a pattern of artificial ventilation designed to produce the optimum

oxygen flux, one way to control end-tidal carbon dioxide concentration ($P_{E'CO_2}$) might be to use a variable deadspace. Dr Fletcher's other comments are well argued, well taken, and add to our knowledge. Having said that, he writes: 'I cannot understand the rationale for the variable deadspace described'.

The project took 18 months, went to 20 drafts, 321 revisions, peer review, the editorial blue pencil and the attachment to an orotracheal tube of a 4.1 kg bespoke brass deadspace (£90.00 plus VAT). Why should we go to all that trouble? The explanation is simple. It was to highlight the last two paragraphs and in particular the very last sentence of our discussion: 'Whether normocapnia offers advantages in the presence of hyperventilation requires further study'.

If we allow a patient to breathe spontaneously under general anaesthesia we are obliged to accept a $P_{E'CO_2}$ of, say, in the range 5.4–7.0 kPa (hypercapnia). When we ventilate the lungs artificially and are thus able to manipulate arterial PCO_2 , one of the few physiological variables the anaesthetist can easily manipulate, we are not obliged to accept any value of $P_{E'CO_2}$; we can select one. Why do we so often select a value less than 4.8 kPa (hypocapnia)? More to the point, does it matter?

Our rationale, then, was to foment controversy. The answers to our questions, if they deserve answers, cannot be had, we are told, without a multicentre controlled clinical trial of such mindbending complexity as almost to preclude meaningful results. Is that true?

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Anaesthetic death of a child with a cold

We report the anaesthetic death of an undersized girl who was recovering from an upper respiratory tract infection (URTI) at the time of her operation. The child was aged 15 months and weighed 6.5 kg. She had had a 'chesty cold' for 2 weeks prior to admission for elective squint surgery. On examination, despite a runny nose, she was lively and afebrile and her chest was clear. The anaesthetist who examined her felt she was fit for anaesthesia. This was induced and maintained with nitrous oxide, oxygen and halothane. She received 15 mg suxamethonium for tracheal intubation. The operation was uneventful and the child breathed spontaneously throughout. Monitoring was by continuous ECG trace and pulse oximetry.

At the end of surgery the child was turned onto her side and her trachea extubated with the monitoring still connected. She made no respiratory effort and was hastily re-intubated. It was noted at this time that she was in asystolic cardiac arrest. External cardiac massage was commenced and several boluses of adrenaline 100 µg were given intravenously. After 25 min a spontaneous cardiac output was restored. There was some movement of all four limbs, but she developed pulmonary oedema. Frusemide 7.5 mg was given and she was transferred to ICU for ventilation of her lungs, but her condition deteriorated and she died 17 h later.

At postmortem the lungs were heavily congested; but there was no sign of a pneumonia. The brain was mildly oedematous. Otherwise all organs were normal. The child's death was caused by a prolonged postanaesthetic cardiac arrest which was probably provoked by laryngospasm as her trachea was extubated. We felt that the child's death

was related to her physical immaturity (her weight was on the third centile) and the recent URTI.

In retrospect, it is debatable whether the child should have been anaesthetised. A recent publication suggests that URTIs in children are not benign with respect to anaesthesia [1]. Another recent article suggests that an anaesthetist may have been negligent for anaesthetising a child with an URTI [2]. URTIs make the upper airways more irritable [3] and hence may predispose to complications such as laryngospasm which can, in rare instances, prove fatal.

We hope that this report will alert colleagues to the potential dangers of anaesthetising very small children with URTI symptoms, even if the child in question is apparently well in itself and the symptoms are minimal.

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Manual ventilation and the Humphrey ADE breathing system

I have just re-read the suggestion by Dorje (*Anaesthesia* 1990; 45: 341) on the Humphrey ADE anaesthetic breathing system. He presents what appears to be a useful modification of the system to permit manual ventilation. However, the recent increase in the system's popularity prompts me to correct some of his observations and suggest an interesting technique for manual ventilation.

His logic to modify the ADE system has been based on the evidence that Mapleson A systems are inefficient for manual ventilation. Indeed, it was initially my own logic since, in my original design of the dual lever ADE system, I included the possibility of manually ventilating the lungs in the D/E mode, while stressing that it was not advisable to do so in the 'A' mode [1]. However, in resolving criticisms concerning the safety and simplicity of the ADE system, I introduced the single lever system [2], knowing that it would only be convenient manually to ventilate the lungs in the theoretically inefficient 'A' mode. As it became more evident that the ADE did not function exactly like the Magill system, I became curious to investigate the behaviour of the ADE during manual ventilation. Independent research had verified my original observations that the ADE system, during spontaneous respiration, functioned significantly differently compared to the Magill system [3] and that the ADE was undoubtedly more efficient and could be used with lower fresh gas flows [4]. Consequently the function of the ADE system during manual ventilation, both in the 'A' and 'D' modes (using my own modification rather similar to Dorje's), was evaluated and compared to the Magill in anaesthetised subjects [5].

Significant differences were observed; the Magill system was confirmed to be inefficient, while the ADE in the 'A' mode was shown to be particularly effective and, even more surprisingly, more so than in the 'D' mode. The average end-tidal carbon dioxide tensions from 10 anaesthetised patients utilising within-patient comparisons when using fresh gas flow of $70 \text{ ml.kg}^{-1}.\text{min}^{-1}$ were 6.8 kPa with the Magill, 4.8 kPa with the ADE in the 'A' mode and 5.2 kPa with the ADE in the 'D' mode [5] (Fig. 1). More practically it was observed that the efficiency of the 'A' mode was preserved despite considerable variations in tidal volume and frequency as would occur during manual ventilation in a clinical situation (Fig. 2). End-tidal carbon dioxide remained almost constant; it appeared that overventilation with expected carbon dioxide washout was compensated by a greater degree of rebreathing of alveolar gases.

These observations again demonstrate that the Mapleson

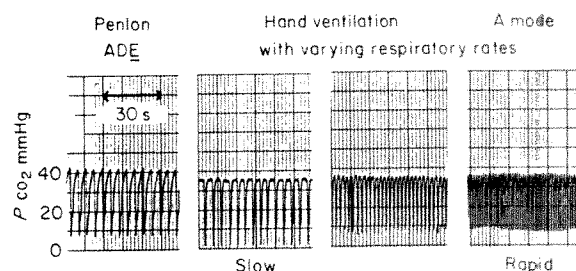


Fig. 2. A typical capnograph trace from one anaesthetised subject comparing manual ventilation with the ADE set in the 'A' mode. FGF was fixed at $70 \text{ ml.kg}^{-1}.\text{min}^{-1}$ and a reference normocarbica value was again obtained during automatic controlled ventilation with the ADE system in its 'E' mode (block 1). Despite a four-fold variation in ventilation volume with slow or rapid rates, end-tidal carbon dioxide tensions did not significantly change. End-tidal carbon tensions are thus fresh gas dependent and ventilation independent.

A system and its modifications do not function alike, and that any modification made to a basic design must be fully re-assessed. More importantly, the differences shown in this instance are beneficial, so much so that I now strongly recommend the use of the ADE in its 'A' mode for manual ventilation. Moreover, end-tidal carbon dioxide tensions are determined by the fresh gas flow and not the ventilation volume. If carbon dioxide build-up is required (as is often the case at the end of an anaesthetic), I use 1 litre of oxygen and 'overventilate'. Oxygenation is ensured while carbon dioxide tension rises. No longer does one have to wait patiently to build up carbon dioxide by hypoventilation, nor is it necessary to have a cylinder of carbon dioxide on the anaesthetic machine.

Thus I conclude that no compromise concerning the inherent simplicity of the design of the single lever ADE system is necessary. In practice, the system is used with the lever up for spontaneous and manual ventilation, and down for controlled ventilation. No detachment of the ventilator or reservoir bag is necessary during any changeover between any of the modes of use.

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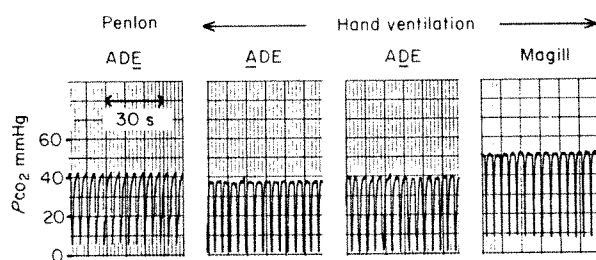


Fig. 1. A typical capnograph trace from one anaesthetised subject comparing manual ventilation with the ADE and Magill systems. FGF was fixed at $70 \text{ ml.kg}^{-1}.\text{min}^{-1}$ and a reference normocarbica value was obtained during automatic controlled ventilation with the ADE system in its 'E' mode (block 1).

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Suprascapular nerve block in the management of cancer pain

We read with interest the article by Wassef describing the use of suprascapular nerve block in the management of frozen shoulder (*Anaesthesia* 1992; 47: 120-4). The use of this block for cancer pain has not been reported previously and we would like to describe its use in the management of shoulder pain due to a solitary metastasis from breast cancer.

A 39-year-old woman presented to the Pain Clinic with a 9 month history of pain in the left shoulder. She had undergone a right partial mastectomy for carcinoma of the breast 21 months previously. A local recurrence had subsequently been successfully treated by radiotherapy. X ray and bone scan showed a single destructive deposit in the head of the left humerus consistent with a metastasis. This had also been irradiated, but was unchanged on a bone scan. The shoulder pain was poorly controlled by escalating doses of morphine, naproxen and mefenamic acid, and the patient was severely troubled by nausea and vomiting produced by these drugs.

Using a standard technique [1], a left diagnostic suprascapular nerve block was performed using 0.5% plain bupivacaine and methylprednisolone. This resulted in complete relief of her pain and she was able to stop taking all analgesics until the following day. Her shoulder pain then returned and she underwent a left suprascapular nerve cryolesion using a Spemby SL 2000 Neurostat with a Lloyd cryoneedle under Image Intensification. Two freeze-thaw cycles were performed, with each freeze timed at 3 min. The patient was pain free immediately after this procedure and required no further analgesics. She remained

free of pain when followed up at 4 months and at 12 months.

Suprascapular nerve block is well recognised in the management of chronic noncancer shoulder pain [2-4]. The use of this block in cancer pain has not been reported previously and should be considered in patients with isolated deposits in the head of the humerus in whom opioids and other drugs are poorly tolerated [5,6].

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Suprascapular nerve block. New indications and a safer technique

We were interested to read the study on the application of suprascapular nerve block for the management of shoulder pain by Professor Wassef (*Anaesthesia* 1992; 47: 120-4). We have not been able to identify a previous description of the block with nerve stimulation as part of the technique and would like to add our own observations.

Our experience of suprascapular nerve block has been in the management of postoperative pain following open or closed (arthroscopic) shoulder surgery, particularly acromioplasty. We were surprised at the efficacy of the block in relieving pain in what is a notoriously painful operative site and we undertook a preliminary study of 11 patients having shoulder arthroscopy with or without arthroscopic surgery of the acromioclavicular joint. The suprascapular nerve was located in anaesthetised subjects using a Braun Stimuplex peripheral nerve stimulator and 5 cm 20 gauge insulated needle. It is not clear whether Professor Wassef used a short bevel needle, but we feel that this is essential to avoid nerve damage. The surface marking for needle insertion was obtained by drawing a line to divide the length of the spine of the scapula into thirds and drawing a second line perpendicular to the first at the junction of the medial third and lateral two-thirds. The scapular notch lies 1 to 2 cm cranial to the point of intersection. The classical method [1] involves drawing a line to bisect the scapular angle; however, bony landmarks are difficult to palpate in obese or muscular subjects and the spine may be more readily identified than the angle.

Professor Wassef mentions that it is not necessary to turn the patient with the anterior approach and this is also true of our posterior approach, when some experience has been obtained. Supraspinatus and/or infraspinatus twitch was observed directly and confirmed by an assistant

supporting the arm with the elbow in flexion, when abduction and external rotation were readily detected. In eight cases the branch to supraspinatus was stimulated and in three, infraspinatus twitch also occurred indicating main trunk stimulation. The nerve was identified at a depth of 1.5-3 cm, which accords with Gordh's description [2]. When muscle twitch was still present at 0.5 mA and abolished by the injection of 1 ml of local anaesthetic, a further 9 ml of 0.5% bupivacaine with adrenaline 1:200 000 was injected. The cutaneous distribution of the suprascapular nerve is a variable feature and complete analgesia may require supplementary subcutaneous infiltration. Occasional difficulty in identifying the nerve may be due to pre-existing rotator cuff rupture and supraspinatus atrophy.

We consider that this technique minimises the risk of complications (pneumothorax and intravascular injection) and provides a simpler and safer alternative to interscalene or supraclavicular brachial plexus block in the management of this type of postoperative pain. We note that Professor Wassef did not include a control group in his study and this must clearly be an essential part of any more formal assessment of the block.

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Intra-articular morphine for the management of frozen shoulder

We read with interest the article by Dr Wassef (*Anaesthesia*, 1991; 42: 120-4). The management of frozen shoulder by sympathetic blockade at the level of the suprascapular nerve is appealing. We propose that treatment at a more peripheral site would also be effective.

Several studies have demonstrated that the synovium is innervated by sympathetic nerves [1,2]. Thus, opioids administered into the shoulder joint should act on the intra-articular fibres of these nerves and provide pain relief in patients with frozen shoulder. We believe that the sympathetic blockade achieved at an intra-articular level may be more specific. Early studies that we have carried out have shown effective results. In our technique, after manipulation of the shoulder under general anaesthesia, 5 mg of preservative-free morphine in combination with 25 ml of 0.25% bupivacaine and 80 mg of depomedrone are injected into the shoulder joint. Patients treated by this technique have had good analgesia of longer duration with better tolerance to physiotherapy. As the suprascapular

nerve contains a high proportion of sympathetic fibres supplying the shoulder joint, we suggest that the response to Dr Wassef's technique could be improved by the addition of morphine.

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Suprascapular nerve block

I read with interest the article on suprascapular nerve block by Wassef (*Anaesthesia* 1992; 47: 120-4) and congratulate him on describing this new approach; it looks promising for the management of shoulder pain.

His article suggests that suprascapular nerve block was first described by Bonica [1] whereas in fact it was reported by Wertheim and Rovenstine in 1941 [2], and its use in the management of shoulder pain was described in 1949 [3].

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Potential hazard with the M&IE Carden Ventmasta ventilator

All safe anaesthetic breathing systems incorporate mechanisms to prevent or limit unduly high airway pressures which can lead to pulmonary barotrauma. Often these take the form of a compliant reservoir bag or pressure relief valve. I should like to describe a case in which these mechanisms were bypassed resulting in a life-threatening complication.

A 31-year-old male suffering from peripheral vascular disease was undergoing a femoropopliteal graft embolectomy. Anaesthesia was administered by a senior registrar using a low fresh gas flow through an M&IE circle system incorporating a Jumbo absorber. The patient's lungs were ventilated using an M&IE Carden Ventmasta ventilator, the inspiratory port of which was connected to the bag mount of the absorber by a piece of black rubber tubing. Approximately 45 min after the start of the procedure, in order to deepen anaesthesia whilst a top-up of muscle relaxant was taking effect, the patient's lungs were briefly hand-ventilated by disconnecting the ventilator end of the interposing tubing and inserting a black rubber bag into it. After he had settled, the bag was removed but the interposing tubing was inadvertently incorrectly connected to the expiratory port of the ventilator. The patient's oxygen saturation began to fall, his chest ceased to move and it was noted that the ventilator bellows failed

to fill. The situation was misinterpreted as a loss of fresh gas resulting from the disconnection and an attempt was made to refill the system from the high pressure emergency oxygen flush of the anaesthetic machine. This resulted in a very high airway pressure being applied to the patient's lungs and the consequent development of a bilateral tension pneumothorax. Fortunately both the error and the complication were spotted very quickly and chest drains were inserted bilaterally. The patient subsequently made a complete recovery and was discharged from hospital 9 days postoperatively.

The misconnection described above is easily made since the inspiratory and expiratory outlets are identical male connectors. This situation conforms to British Standard BS 3849:1988. When erroneously connected as described above, there is no pressure relief device in the system as the bag outlet of the absorber is connected directly to the spool valve housing of the ventilator which is closed to the atmosphere. The spill valve of the circle system has to be completely closed when using this ventilator with low fresh gas flows and the reservoir bag is not incorporated in the system.

Following discussion with the anaesthetic department and with the manufacturers it was decided to incorporate a 60 cmH₂O pressure relief valve into the breathing system

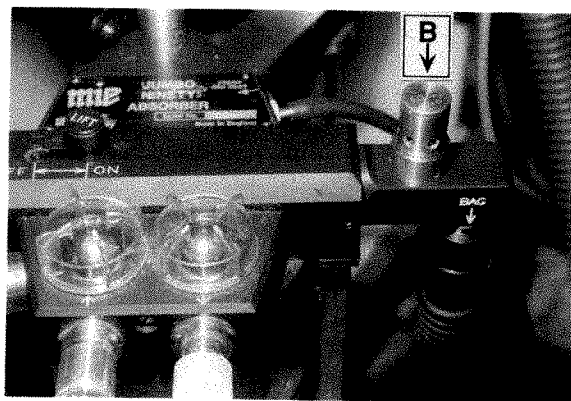
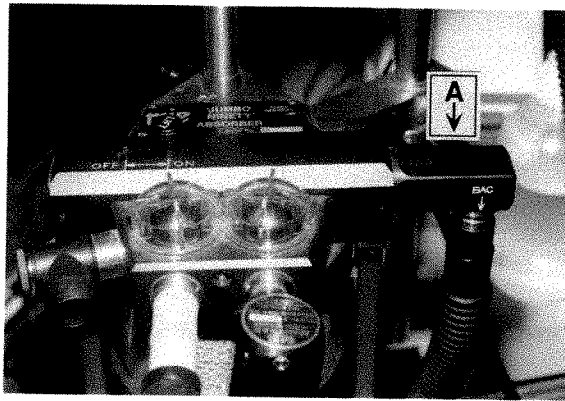


Fig. 1(a). The unmodified M&IE Jumbo absorber system showing the bag mount (A) and the interposing tubing connected to it. **(b)** The modified system showing the 60 cmH₂O pressure relief valve (B) on top of the bag mount.

(Figs 1(a) and (b)) to limit overpressure from any source. The manufacturers themselves have also modified both the Carden Ventmasta ventilators at this hospital by placing a fenestrated plug into the end of the spool valve housing, so that this is open to the atmosphere and will produce a

whistle if a misconnection occurs. Both modifications were carried out promptly and free of charge by M&IE.

It would seem prudent for all anaesthetic departments which use this system to consider similar modification of their apparatus in the interests of safety. The problem has been reported to the Department of Health.

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N.M. TURNER

A reply

Thank you for the opportunity to reply to the letter from Dr N.M. Turner, regarding the problem he had when inadvertently misconnecting the Carden Ventilator.

Most incidents are caused when, not one, but a series of mishaps happen as in this case. Firstly the tubing connecting the ventilator bellows to the absorber was removed to manually ventilate the patient's lungs even though a separate manual ventilation system is supplied with the Carden. Secondly, after manual ventilation the tubing was wrongly connected to the expiratory port instead of the inspiratory port as they are both male 22 mm tapers complying with BS 3849:1988, although clearly marked 'INSP' and 'EXP'. Thirdly, the deflated bellows was mistakenly interpreted as a lack of fresh gas.

As Dr Turner states, we were able to fit a 60 cmH₂O relief valve to the circle absorber system the same day that the incident was reported to us. The following day an even more elegant solution to this problem was considered and that was to replace a solid end cap with a fenestrated plug, which is open to atmosphere should the connecting tube be connected to the expiratory port, by mistake, when the switch is in the Mapleson D/E/Absorber position.

I can confirm that we have already modified, free of charge, all the switchable Carden Ventilators that are in use to prevent a repeat of this incident.

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T. LONGMAN

No more complications with stylets

We read with interest the letter from Martin and Campbell (*Anaesthesia* 1992; **47**: 75-6). We have knowledge of an instance when the tip of a stylet broke off and entered the trachea, but was fortunately coughed up the next day. We know of a further case, albeit 25 years ago, where a stylet actually perforated the trachea resulting in a pneumomediastinum.

We have abandoned the use of the stylet to aid tracheal intubation and have been pre-shaping the tube by immersing it in ice cold water [1]; consequently all problems associated with a stylet are avoided. Our method cannot, however, be employed with armoured tubes which

are made of silicone rubber. Recently, armoured tubes made of PVC have been introduced where our method can be employed.

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Reference

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A badly fitting tray

During a routine teaching session on the anaesthetic machine we removed the metal tray on a B.O.C. Boyle International Mark 2 to look at the gas arrangement below. On attempting to replace the tray we found this

difficult, as the slots cut into the tray for the pipes up to the Rotameters did not align correctly and caught upon the oxygen pipe. On looking further it was noticed that obviously, over a period of time, the tray had started to



Fig. 1.

'saw' into the oxygen pipe, as shown in the photograph. The machine was removed from service and Ohmeda informed to replace the pipe and enlarge the slots in the tray.

On checking all International Mark 2s we found evidence of slight damage to a pipe on another machine

and would recommend all machines are checked for alignment and pipe damage.

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K. NATHAN
D. PERKS

A reply

Thank you for the opportunity to comment on the letter from Drs Nathan and Perks. This is the second report of this nature we have received since introducing this product in the early 1970s. Neither of these reports indicate that gas leakage has occurred resulting from this abrasion to the pipework.

The degree to which the pipes may become damaged will obviously be influenced by the age of the machines and the care with which the tray has been removed and replaced. The International II was discontinued in 1985, but is still supported by Ohmeda Service. Those machines which are on Service Contract with Ohmeda are subject to a routine inspection of the pipework with resultant replacement if necessary.

Any users having concern regarding the integrity of the gas pipework on any of their Ohmeda machines should contact the Technical Support Department at Hatfield on 0707 263570 ext 4003 with the model and serial number of the equipment concerned.

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R. DAVIES

Blood reflux, central venous cannulation and right-sided haemothorax

We read with interest the case report by Pina, Morujao and Castro-Tavares (*Anaesthesia* 1992; **47**: 30-1) concerning unreliable signs of correct position of an internal jugular catheter in a patient with thoracic trauma. We also wish to report a patient in whom reflux of blood, respiratory fluctuations and even chest radiography wrongly indicated correct placement of a subclavian catheter in a patient with thoracic trauma and haemothorax.

A 35-year-old female patient was admitted to the emergency room following attempted suicide by jumping off a bridge. She had multiple fractures of the pelvis, right

humerus and ulna, 9th thoracic vertebra and ribs (6-10), with a right-sided haemothorax (Fig. 1). The right subclavian vein was cannulated using the infraclavicular approach and Seldinger's technique. A 7 FG Shaldon catheter was inserted, from which blood was withdrawn for analysis and cross-matching. After insertion of a chest drain and drainage of 1500 ml of blood, a further chest X ray showed complete resolution of the haemothorax and apparently correct placement of the catheter (Fig. 2).

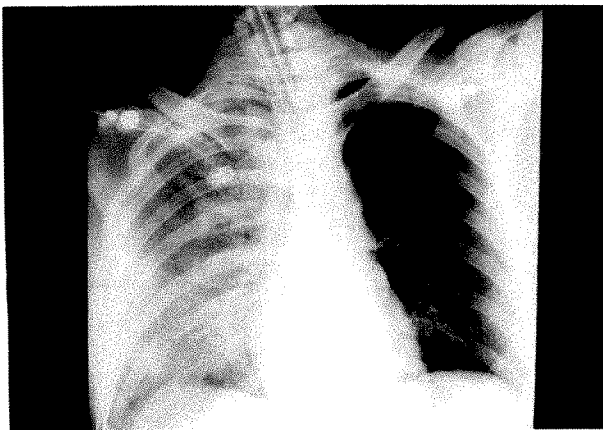


Fig. 1. Chest X ray taken immediately after admission to the hospital showing a right sided haemothorax.

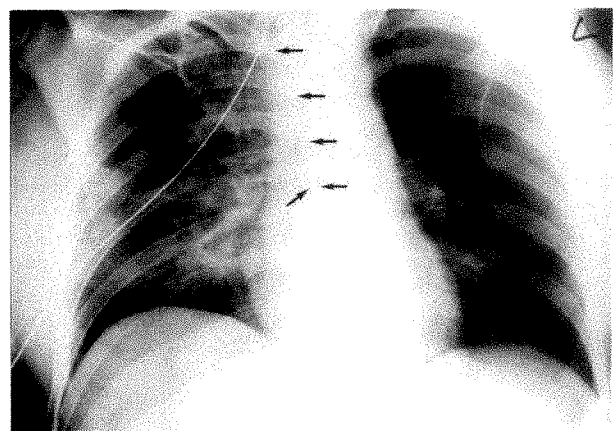


Fig. 2. Chest radiograph following insertion of a Shaldon catheter via the right subclavian route and successful drainage of the haemothorax. The arrows mark the shape of the incorrectly placed catheter and the position of the catheter tip.

However, despite continuous volume replacement, her blood pressure deteriorated and drainage of dark blood from the chest drain increased. Laceration of a major vessel was suspected and two additional cannulae were inserted into the right internal jugular and right femoral veins. Right thoracotomy revealed bleeding from intercostal vessels and the tip of the Shaldon catheter lying in the pleural cavity. As only blood had been administered through it, the colour of the drained fluid did not raise suspicion. The result of the coagulation tests (PTT and thrombin time >180 s, prothrombin time $<1\%$) indicated that the initial blood sample had come from the haemothorax (a second sample taken from the internal jugular vein showed no evidence of a coagulopathy). Following coagulation with electrocautery and withdrawal of the subclavian catheter, the chest was closed; the patient made an uneventful recovery.

We agree with the authors that in patients with thoracic

trauma, reflux of blood and respiratory fluctuations do not prove correct catheter placement. As our case illustrates, even radiological control may be misleading. We suggest that in patients with thoracic trauma and a haemothorax, central venous catheter insertion should be performed with X ray control and with injection of contrast medium, which will spread into the pleural cavity in case of malposition of the catheter. Failure of clot formation in an initial blood sample should raise suspicion that the catheter is sited in the pleural space.

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Reversal of prolonged suxamethonium apnoea

In their paper on the use of fresh frozen plasma (FFP) to treat plasma cholinesterase deficiency in an infant, Gill, O'Connell and Scott (*Anaesthesia* 1991; 46: 1036–8) quote Scholler *et al.* [1] as having used bank blood as a source of plasma cholinesterase. However, Scholler *et al.* did not actually use blood as treatment for prolonged apnoea, they merely observed that peri-operative blood transfusion could mask a pre-existing plasma cholinesterase deficiency. The only report which I can find of the use of blood bank to treat plasma cholinesterase deficiency is that of Garry *et al.* [2], who used 2000 ml of whole blood in a patient homozygous for the silent gene and determined that the half-life for the transfused plasma cholinesterase was 3.4 days.

I am interested that the authors consider that the relative risk associated with administration of fresh frozen plasma (FFP) changes with time, as they seem to imply in their final paragraph. Whatever the perceived risk of FFP administration, this risk is constant. It is for the individual to decide whether FFP provides a safe source of plasma cholinesterase, but there is no justification for altering the opinion merely because time is passing. In the UK the chance of disease transmission from a unit of FFP is 'negligible' (Scottish National Blood Transfusion Service 1991; Crawford RJ, personal communication), but the risk

associated with continuing anaesthesia and ventilation for this otherwise healthy infant in a postoperative high dependency unit is also extremely small. The case report was interesting, but where facilities exist for safe artificial ventilation, this must remain the preferred treatment option for prolonged apnoea after suxamethonium [3]. Since each unit of FFP is derived from a single donation, and not a small donor pool, it is also worth remembering that one unit of FFP carries a 0.03% risk of coming from a donor who also has a plasma cholinesterase deficiency.

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And where does the time go during operating lists?

I was interested to read the recent letter 'And where does the time go during operating lists?' by J. Turner (*Anaesthesia* 1992; 47: 69–70).

Whilst accepting that a considerable variation in 'induction time' exists between individual anaesthetists, I question whether the 22%–46% variation in 'nonoperating time' quoted as occurring at Dunfermline and West Fyfe Hospital is always *principally* the result of this factor. Like the writer, I would not presume to cast aspersions on another's working practice, but no specific reference is made to the interval between the anaesthetised patient being placed on the operating table, and the surgeon commencing the skin cleansing. Why are surgeons (in general) so reluctant to start scrubbing up at the same time as the anaesthetist (unless he/she is very slow) starts the

induction? One of the principal causes in my experience, of delay in 'getting on with the operation' is the futile time spent in persuading the surgeon to come back into the operating theatre when the patient is already waiting on the table. A 10 min wait is not at all uncommon; it does not require a mathematician to deduce that it takes only six such patients to add another hour's operating time to the list. But perhaps north of the border our surgical colleagues are better disciplined, and also if I had the pleasure of anaesthetising for a woman surgeon I would not be writing this reply!!

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J.G. FRANCIS

Epidural infusion of bupivacaine and diamorphine for postoperative analgesia on surgical wards

We were delighted to read the paper by Hobbs and colleagues (*Anaesthesia* 1992; 47: 58–62) on the value of epidural infusion combining diamorphine and bupivacaine for postoperative analgesia, particularly in the clinical environment where pulse oximetry and capnography monitoring are not readily available. The wider availability of such quality analgesia should be encouraged with appropriate education of nursing staff and supervision by an acute pain control team, as recommended in the Joint Working Party on Post-operative Pain [1]. However, we would like to make two comments, the first regarding the incidence of respiratory depression, quoted as 6%, occurring up to 24 h after the beginning of the analgesic infusion.

In our series of 800 patients receiving intermittent boluses of epidural diamorphine (2.5 mg in 10 ml saline) [2] only seven instances (0.9%) of respiratory depression occurred, defined in our study as a respiratory rate of less than 10 breath.min⁻¹; five of these occurred in the recovery ward within an hour of the first dose of epidural diamorphine. Of the remaining incidents, one, in a patient with obstructive airways disease, took place during weaning from IPPV in the intensive care unit; in the other there was a breach of protocol and 5 mg diamorphine was given. The loading dose reported in Hobbs' paper of a mean value of 2.0 mg is roughly equivalent to our first dose and this is then followed by an infusion, tailor-made to the patient and the nature of the surgery, to a mean of 9.5 mg diamorphine per 24 h in bupivacaine solution. Despite a relatively lower limit as a definition of respiratory depression, we were not surprised to see that this represented a significant side effect in their study, when the synergism of opioid and local anaesthetic has to be taken into account. We accept that respiratory depression might occur up to 6 h after an epidural top-up of diamorphine; however, our experience shows that in clinical practice this is not so, but is more likely to occur in the recovery room after the first dose has

been given, and demonstrably when intravenous opioids have been used preoperatively. There was no mention in Hobbs' paper of the duration of observation of the patient in the postoperative recovery ward, which we feel should be an important aspect of epidural management.

Secondly, it was of some concern to find that the ward staff were encouraged to adjust the infusion rate. If analgesia was found to be insufficient, this surely would be better corrected by the administration of a bolus dose followed by an increase in the infusion rate, thereby avoiding a prolonged latent period for analgesia to become effective? Furthermore, we feel that such a procedure would be better supervised by a member of the medical staff or one of the acute pain control team.

Whilst it must be appreciated that no technique of postoperative analgesia will be 100% safe, as anaesthetists we should be striving to develop the highest efficacy/safety ratio for postoperative pain relief, and in this respect, Hobbs *et al.* are to be applauded. We suggest, however, that a technique which incurs a rate of respiratory depression of 6% with potentially delayed onset is unacceptably high.

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Fibreoptic laryngoscopy as a method of assessing the risk of airway obstruction following laryngeal mask airway insertion

Downfolding of the epiglottis is the commonest cause of airway obstruction following laryngeal mask airway insertion [1,2] and appears to be commoner in children [3]. However, even under these circumstances, clinically the airway is usually patent [4].

We performed, under local anaesthesia, nasal fibreoptic laryngoscopy in 24 consecutive ASA 1 or 2 patients presenting for elective gynaecological surgery in whom a laryngeal mask (LMA) was to be used, in order to estimate the risk of possible downfolding of the epiglottis. The size of the epiglottis, its position (anterior or posterior) and the size of the aryepiglottic folds were noted. Anaesthesia was then induced with propofol and fentanyl and an LMA (size 3 or 4) passed in the recommended manner by an anaesthetist who was unaware of the laryngoscopic findings. Once *in situ*, laryngoscopy was repeated via the LMA to confirm its position.

In eight of the 24 patients, the airway was obstructed by the epiglottis, this being partial in seven and complete in one. The main association with the airway obstruction was a posteriorly placed epiglottis as seen at the original laryngoscopy. A posteriorly placed epiglottis was found on 11 occasions and seven of these were associated with airway obstruction. A normal epiglottis obstructed the airway on one occasion only. This difference was

significant ($p > 0.05$, chi-squared test). There were no cases of obstruction due to large aryepiglottic folds or a large anteriorly placed epiglottis.

These results indicate that a posteriorly placed epiglottis is a risk factor for airway obstruction when using a LMA and that this can be predicted by nasal fibreoptic laryngoscopy. However, as most of the cases of obstruction are partial, fibreoptic laryngoscopy will not be necessary in the majority of instances [5], but it could give useful information if tracheal intubation through a LMA is contemplated [6].

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Patient misunderstanding

I notice with interest the paper by Saunders *et al.* (*Anaesthesia* 1991; **46**: 1056-8) which suggests that, despite firm convictions about correct medical appearance, patients either did not observe, or did not care, how their anaesthetist was dressed. I am afraid that patient misunderstanding is deeper than this.

I recently interviewed 50 patients (mean age of 57 years) who were visited between 1 and 7 days after orthopaedic or general surgery. The survey was performed in a University Teaching Hospital, and included patients who had undergone both elective and emergency surgery. When asked for the name of the consultant surgeon in charge of their care, 36 (72%) gave the correct answer, six (12%) did not know, and eight (16%) thought that they knew, but named the wrong person. Only 25 (50%) knew who had performed their operation. All patients believed that the person who performed their operation was a specialist or an ordinary doctor, none thought that it had been performed by a nurse or orderly. Although 78% of the patients thought that an anaesthetist had visited them before the operation, only one could name their anaesthetist. Eleven (22%) of the patients thought that a nurse or an orderly had administered their anaesthetic although at least six of these patients had been visited by the anaesthetist before the operation.

I also asked the patients to describe briefly the age and sex of the person administering their anaesthetic. Seven (14%) of the patients got the sex of their anaesthetist wrong. This mistake appeared equally likely with male and female anaesthetists. It is probable that these patients mistook one of the nurses or ODAs for the person who administered their anaesthetic. Overall, 24% of the patients either thought that their anaesthetist was an orderly, a nurse, or totally mistook the identity of their anaesthetist. A pre-operative visit by the anaesthetist did not prevent this from occurring.

On admission to hospital patients meet many new people. They may be so overwhelmed by this that they become unable to distinguish between hospital staff members. A substantial number of people must leave hospital believing that they have been anaesthetised by a nurse or orderly. This must affect the profession's public standing, and may contribute to ill-feeling among patients. I would be interested if colleagues have any suggestions how to remedy this problem.

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Decorative hairbands—an unusual cause of difficult intubation

A 31-year-old female, ASA Grade 1, presented as a day case for laparoscopy. She had been anaesthetised on two previous occasions: in 1988 she had a laparoscopy and it had been noted that an introducer was required to aid tracheal intubation with an 8 mm tube. Two years later she was anaesthetised for tubal surgery, and a size 3 laryngeal mask airway was used successfully.

On the present occasion she was assessed pre-operatively and was not found to have any features which might suggest that intubation would be difficult. Anaesthesia was induced with propofol, and after ensuring that her lungs could be ventilated easily with a face mask, she was given atracurium 30 mg. Ventilation continued with nitrous oxide, oxygen and halothane until muscle relaxation was adequate. At laryngoscopy, however, only the tip of her epiglottis was visible. A gum elastic bougie was introduced and an 8.0 mm tube was passed over this, but the tube was misplaced in the oesophagus. Repeat laryngoscopy, after a further period of ventilation, again revealed only the tip of the epiglottis despite repositioning the head and neck. At this point the patient's operating theatre cap was removed, to reveal that her hair was being kept in place by a decorative hairband, producing a very prominent outcrop of hair (Fig. 1). The hairband was removed, allowing her hair to fall down freely, and laryngoscopy was repeated, this time allowing a clear view of the vocal cords. Her trachea was then intubated easily.

It is concluded that this commonly used hair decoration was the cause of unexpected difficulty in visualising the larynx by limiting extension of the head in the supine position. It is therefore suggested that such potentially hazardous items should always be removed before the



Fig. 1.

patient reaches the anaesthetic room, and should not be allowed to remain hidden under operating theatre caps.

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Making up inotrope solutions

Rooney proposed a formula to minimise drug wastage when delivering inotropes via a syringe pump (*Anaesthesia* 1992; 47: 83). His formula gives a final volume of solution from a known drug dose and patient body weight, which will then deliver the drug at a concentration such that $1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1} = 1 \text{ ml} \cdot \text{h}^{-1}$. I would like to suggest a much simpler solution. This gives final volume (ml) = $0.25 \times \text{drug dose (mg)}$. Body weight is excluded. This simple equation can be used for all adults, although $1 \text{ ml} \cdot \text{h}^{-1}$ will deliver precisely $1 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$ for a 66.7 kg patient. However, inotropes are not strictly administered as predetermined doses according to precise body weights, but are instead titrated to the clinical response. The requirements of dobutamine or dopamine in adult patients

usually range from $2\text{--}15 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$, and this delivery range of $2\text{--}15 \text{ ml} \cdot \text{h}^{-1}$ allows easy adjustments. Also, since most patients who require inotropes are not weighed daily (or perhaps even at all), body weight equations are imprecise anyway. The above simple equation will also work quickly 'in reverse' i.e. derive the dose needed to make up a solution of required final volume, since the ratio of final volume (in ml) to dose (in mg) is always a simple 1 to 4.

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T.E. OH

Change to the TVX transducer cartridge

I would like to report a change that Ohmeda have agreed to make to the black adaptor that is supplied with the TVX Transducer Cartridge for use with Excel ventilators.

The cartridge is supplied with a small black adaptor to fit to the 15 mm connection of a tracheal tube, as shown in Fig. 1. A problem is caused by the fact that this black adaptor had a 22 mm knurled outer rim, that will fit tightly into a 22 mm female connection, as shown erroneously fitted into a catheter mount in Fig. 2. However, because of the knurled edge, a leak is inevitable, which is not audible above other noises, but is apparent when inspired and

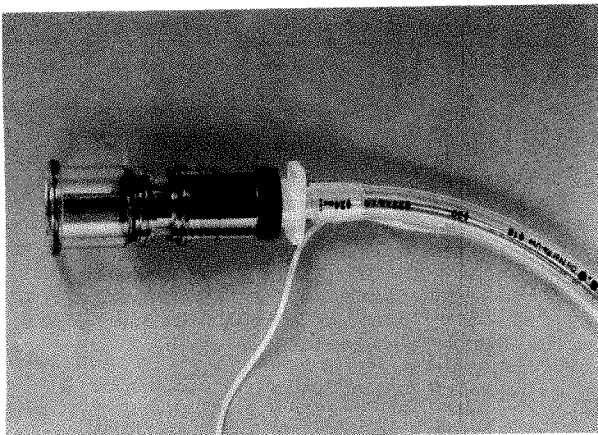


Fig. 1. TVX Transducer Cartridge with black adaptor correctly fitted to 15 mm tracheal tube connector.

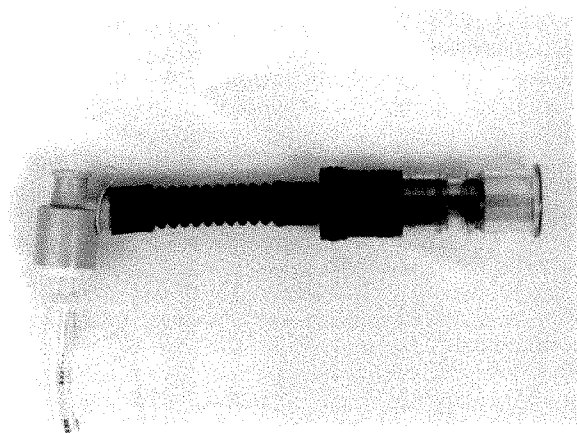


Fig. 2. TVX Transducer Cartridge with black adaptor wrongly fitted into 22 mm female connector.

expired volumes are compared; this emphasises the value of always measuring the expired volume. The adaptor will, in future, be made with an outside diameter greater than 22 mm and the knurled portion will incorporate small thumb grips for fitting and removing. In the meantime I would suggest care in ensuring that the present adaptor is not fitted into 22 mm connections.

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G. ARTHURS

A difficult airway managed by computer?

The introduction of a computerised infusion system to deliver propofol [1] has allowed anaesthetists to alter the depth of anaesthesia in a simple and controlled manner. We would like to report a case where we used this system to manage a patient with a difficult airway.

A 24-year-old female presented as an emergency for exploration of a blocked ventriculo-peritoneal shunt. She had signs of raised intracranial pressure. Pre-operative examination revealed an obese woman of below average intelligence, with spina bifida, hydrocephalus, and a fixed

flexion deformity of her cervical spine. Her old notes were not available. We therefore treated her as a potentially difficult tracheal intubation. Venous access was established and 100% oxygen administered via a Bain system. Continuous monitoring of heart rate, blood pressure and oxygen saturation was instituted. Anaesthesia was induced with propofol over a 10 min period using the computerised system by increasing the target concentration in $0.5 \mu\text{g} \cdot \text{ml}^{-1}$ increments. A laryngeal mask was inserted without difficulty at a target concentration of $4.5 \mu\text{g} \cdot \text{ml}^{-1}$

and anaesthesia maintained by the propofol infusion ($6.0\text{--}3.0\ \mu\text{g}\cdot\text{ml}^{-1}$) with the patient breathing oxygen-enriched air. This was supplemented by the surgeon using local anaesthetic infiltration. Exploration of the distal end of the shunt was not required and the operation lasted 30 min. Oxygen saturation remained about 95% throughout and the highest recorded end-tidal CO_2 was 6.5 kPa. The patient made a swift and uneventful recovery.

We would like to recommend this technique of controlled intravenous induction and maintenance of anaesthesia as a possible alternative in patients with

difficult airways in whom the use of a volatile agent is contraindicated.

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Can calcium channel blockers prior to tracheal intubation prevent myocardial ischaemia?

I read with interest the article by Omote *et al.* (*Anaesthesia* 1992; **47**: 24–7). I would like to make a few comments regarding the clinical implications derived from their investigation. Their article demonstrated that nicardipine $20\text{--}30\ \mu\text{g}\cdot\text{kg}^{-1}$ prevented effectively the increases in blood pressure and rate-pressure product associated with laryngoscopy and tracheal intubation in hypertensive patients, although they observed no significant difference in heart rate responses to tracheal intubation among the patients with and without nicardipine pretreatment. These results are consistent with previous investigations [1,2] showing how little vasodilators affected heart rate increases following tracheal intubation.

Based on their present results and other reports regarding the effects of nicardipine on myocardial metabolism and coronary haemodynamics [3,4], they have assumed that nicardipine seems to be an appropriate agent for attenuation of haemodynamic alterations associated with laryngoscopy and tracheal intubation in patients with cardiovascular disease. However, they have shown no data demonstrating a lower incidence of myocardial ischaemia in the patients receiving nicardipine pretreatment. It is generally supposed that patients with ischaemic heart disease can tolerate hypertension better, and conversely can only tolerate poorly hypotension and tachycardia. Therefore, it seems unlikely that calcium channel blockers are of value in patients with ischaemic heart disease, because their administration prior to laryngoscopy and tracheal intubation may elicit profound hypotension which comprises coronary blood flow with a risk of developing myocardial ischaemia [2].

In addition, Lieberman *et al.* [5] have noted that myocardial ischaemia occurred with significant increases in heart rate, and with significant decreases in blood pressure

and coronary perfusion pressure in patients undergoing halothane anaesthesia for coronary artery revascularisation. They concluded that rate-pressure product was not a useful determinant for predicting myocardial ischaemia. Nevertheless, I believe that further clinical studies should be performed to evaluate whether calcium channel entry blockers could ameliorate myocardial ischaemia in patients with ischaemic heart disease undergoing laryngoscopy and tracheal intubation.

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Blease Manley 4 ventilator and PEEP

I would like to bring to your attention a possible pitfall which may have serious sequelae if unrecognised. It concerns the Blease Manley 4 ventilator which may be used in a semi-closed mode and which allows ventilation via either a Bain system or a circle absorber. Using this mode of ventilation, respiratory frequency is controlled by a separate rate control knob on the top of the ventilator which is ungraduated and if inadvertently turned fully clockwise may stop the ventilator. This, of course, will normally not go unrecognised and will be promptly corrected with no harm to the patient. However, I have recently discovered that if the ventilation is so turned off one of two possible sequelae may ensue, depending upon the phase of respiration. Firstly, if ventilation is on the expiratory phase of the cycle the fresh anaesthetic gas will be spilled in the normal fashion with no deleterious effect to

the patient. The second possibility is that the ventilator is turned off during the inspiratory phase of the cycle. If this occurs, there is not only a maintenance of the PEEP which is applied directly to the patient's airway, but the PEEP will rise *pari-passu* with the fresh gas flow to the circle absorber. This pressure will rise to a maximum of around $70\ \text{cmH}_2\text{O}$, at which point a pressure relief valve inside the ventilator is activated. One can easily imagine therefore the potential horrendous respiratory and cardiovascular consequences which can ensue in only a matter of seconds if the correct circumstances combine.

Nowadays one would anticipate that most modern operating theatres are equipped with ventilation pressure alarms which can alert one to unexpected changes in airway pressure, but unfortunately these are not always in routine use and I would therefore suggest that extreme

vigilance is exercised when using the ventilator in this mode. I would further suggest that the ventilator be modified in such a way as to make such a dangerous build-up in pressure in the patient circuit impossible.

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B. SWEENEY

A reply

Dr Sweeney correctly mentions that it is possible to stop the ventilator using the flow control knob, although he also

correctly points out that this will never go unrecognised. We are aware of the second possibility that Dr Sweeney created; however, we feel that this could only occur when the flow control knob is used incorrectly as a method of turning off the ventilator, and only during the inspiratory phase. This would not typically occur, since the established and accepted method for turning off any Manley ventilator is to disconnect the gas supply.

Should any user feel that their ventilator needs special adaptation to prevent the flow control knob being used to switch off the ventilator, Blease will be happy to assist.

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G. ABBOTT

Maximal respiratory pressure measurement

The concept of measurement in medicine and anaesthesia is to be encouraged, so the paper by Smith and Royall (*Anaesthesia* 1992; 47: 144-5) deserves attention, but it should be put in context, both historically and clinically.

In 1844, Mr John Hutchinson measured maximal inspiratory and expiratory pressures with a mercury manometer in nearly 1200 men of different classes [1,2]. Maximum inspiratory pressures ranged from 1.5 to 7 inches of mercury (52 to 242 cmH₂O) which he graded from 'weak' to 'very extraordinary'. Results were standardised for height, and a nose piece rather than a mouthpiece used 'as the tongue and cheeks produce a power about seven times greater than the true inspiratory muscles' [2]. A portable version was suggested, while a description of an early bell spirometer and the results obtained with it were also presented. The authors of the present report tell us little of the precision, bias, reproducibility and test standards used for their device; in addition no normal values or reproducibility data for their device in man are mentioned.

The application of maximal respiratory pressures to clinical practice is limited by the wide range of values quoted for normal subjects [3-9], making a confident diagnosis of respiratory muscle weakness based on a single reading difficult. Serial measurements might be of great benefit in charting weakness in the individual, if the test were reproducible. The coefficient of variation for repeated measurements over 2 to 5 days' testing in normal subjects is at least 6% to 10% [4,5,8]. Long-term reproducibility may be even worse.

In a study of the long-term reproducibility of maximal inspiratory mouth pressure (PI_{max}) in six normal subjects, increases of +17% to +61% in PI_{max} were seen over 46 to 113 days in all but one subject (an experienced respiratory physiologist) [10]. PI_{max} was recorded in the seated position from functional residual capacity using noseclips and a mouthpiece connected by rigid plastic tubing to a Validyne pressure transducer calibrated with a mercury manometer over the range 0 to -200 cmH₂O. No attempt at respiratory muscle 'training' was attempted, and PI_{max} ranged from -76 to -208 cmH₂O. There was no correlation between the number of observations and the magnitude of the increase in PI_{max}, suggesting that the increases were largely due to a learning effect for the measurement procedure itself, rather than due to a change in the strength of the respiratory muscles. Indeed it has been suggested that in order to obtain a reproducible PI_{max} in patients with chronic airflow limitation, a minimum of nine technically acceptable maximal mouth pressure manoeuvres should be performed [11].

While repeated measurement of maximal respiratory pressure is likely to detect gross change within the

individual, being a voluntary manoeuvre requiring maximal effort, the result will always be subject to patient cooperation and enthusiasm. A single measurement, however, is unlikely to permit the diagnosis of respiratory muscle weakness with any confidence. Respiratory muscle weakness can more usefully be quantified using pressure measurements during a maximal sniff, or following phrenic nerve stimulation, even if these procedures are minimally invasive [12].

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Determination of protamine dose

I read with interest the paper by Keeler *et al.* (*Anaesthesia* 1991; **46**: 925–8) concerning a method for determining the dose of protamine required at the end of cardiopulmonary bypass (CPB). This appears to be elegantly simple and since in this centre we use an empirical regimen, I felt it worthy of further study in relation to our practice.

Closer examination, however, reveals a number of flaws: firstly the technique they describe uses only two activated clotting time (ACT) measurements and assumes a linear dose–response in order to estimate protamine dose. The original work on which they base their technique [1] utilised multiple ACT measurements producing a dose–response curve which, as the original authors pointed out, allowed accurate determination of the dose even when the value was between two of the chosen titration points. Secondly, it has been our observation that individual ACT measurements may vary widely, especially in the range above 500 s. Thirdly, the method used to calculate blood volume in these circumstances is unproven and again at variance with that used in the original paper: this may have led to overestimation of the dose required. Finally the assumption that the calculated dose was, in fact, the minimum effective dose is not validated: the authors only demonstrate that both a very high empirical dose regimen (mean 6.2 mg.kg⁻¹) and a lower calculated dose regimen (mean 4.5 mg.kg⁻¹) adequately reversed heparinisation.

These analytical flaws notwithstanding, the technique, if validated, retains the advantage of simplicity and could potentially reduce the adverse effects of protamine under or overdosing [2,3]. As such it would be of considerable interest.

Inspired by their paper, I undertook a review of 30 consecutive patients presenting at this centre who required CPB. Our regimen for heparin administration is similar to that of Keeler *et al.* (300 IU.kg⁻¹ initially and supplementary doses to maintain ACT > 400 s), but only 3 mg.kg⁻¹ of protamine is given at the end of CPB. Further small aliquots of protamine are administered if the ACT or thrombin time (which is also measured routinely) remain prolonged. Four patients for whom complete results were not available were excluded.

This revealed a mean post CPB-ACT to pre CPB-ACT ratio of 1.03 (SD 0.13, Range 0.86–1.23) and a mean thrombin time of 11.0 s (SD 0.98, range 9.4–13.4 s, where < 12 s is considered normal). Blood loss was considered acceptable in all cases, no blood products being required. No patients were given additional protamine, hence mean dose was 3 mg.kg⁻¹.

These results are comparable to those of Keeler *et al.* and superficially would seem to indicate that an even lower average dose of protamine can be used successfully as part of an empirical dose regimen. However, the group studied is again small and the potency of the protamine used here may be different. I would suggest that a further study is indicated incorporating the following features: a control dose of less than 3 mg.kg⁻¹, a validated method of calculating blood volume and correlation of the calculated concentration of protamine with a direct measurement of resultant concentration.

Much has been written concerning the appropriate dose of protamine after CPB and several titration techniques described [1,3,4]; however empirical regimens continue to predominate [3]. Whilst the ideal remains only to administer the minimum effective dose of all drugs and especially those with a narrow therapeutic window, any new technique must have demonstrable benefits in terms of reduced morbidity and mortality compared with the alternatives in order to gain wide acceptance.

Unfortunately Keeler *et al.* fail to deliver; even worse, their results suggest the question 'Protamine—any need for titration?'

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A reply

We appreciate Dr Train's interest in our study. He raises a few points about the method. Our study was designed to assess objectively the Hemochron Protamine Dose Assay, of the international Technidyne Corporation, USA, as a convenient method for protamine titration to neutralise heparin, compared with an empirical dose regimen. The development of the Hemochron Protamine Dose Assay has been based on protamine dose–response curves. These demonstrate that along two points the dose–response curve correlates quite well with linear dose–response calculations ($r = 0.95$) [1]. The verification of heparin reversal, which was performed 5 min after protamine administration, enabled us to detect if the protamine dose was inadequate. As explained in our methods, a further neutralisation line then enabled us to calculate the additional dose of protamine if it was required. This way the minimum dose of protamine was administered.

Individual ACT measurements do indeed vary in our experience when prolonged beyond 600 s. However, they do not vary widely. Also, when heparin is reversed, the ACT values do lie close to each other.

The calculation of the patient's blood volume is based on a height/weight nomogram [2]. The nomogram has been used effectively by LaDuca *et al.* [1] to calculate the protamine dose and is incorporated in the method used to calculate the patient's protamine dose by the Hemochron Protamine Dose Assay. We think that this must be a better way of assessing a person's blood volume instead of assuming that everybody has a blood volume of 80 ml.kg⁻¹ body weight.

We are not surprised that the empirical protamine dose of 3 mg.kg⁻¹ reversed the heparin in the patients studied by Dr Train. He may inadvertently have been administering too much protamine to some of the patients, depending upon the relative potency of the protamine used. In the titrated protamine dose group, the protamine requirements in half the patients studied were around 3 mg.kg⁻¹, a dose found to be adequate even when we assessed heparin reversal after the patient was admitted to the Intensive Care Unit.

We do not feel that another study, as suggested, would

be useful. Overdosage or underdosage will occur in a significant percentage of patients if an empirical protamine dose is used. An empirical dose of 6 mg.kg^{-1} , as used in our study, may be on the high side, but this was the dose used routinely in our unit prior to the study. However, even higher doses up to 12 mg.kg^{-1} have been recommended in the past [3,4]. Direct measurement of plasma protamine may only serve to confirm that the circulating heparin is reversed.

Subsequent to the completion of our study, we have used the Hemochron Protamine Dose Assay in more than 400 patients. About half the patients needed almost 3 mg.kg^{-1} protamine, but there is a significant variation between individual patients' protamine requirements. While empirical dose regimens appear attractive due to their simplicity, it must be better to assess the proper dose by a relatively easy method.

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Antireflux valves in intravenous opioid analgesia

Drs Turner and Murphy (*Anaesthesia* 1991; 46: 1064-5) question whether antireflux valves are necessary when a pump-driven opioid infusion is being 'piggy-backed' into a gravity-driven intravenous infusion line. An episode of significant reflux has occurred in this hospital when a cannula became blocked. The antireflux valve had been placed incorrectly, thereby protecting the opioid infusion (patient controlled analgesia) instead of the gravity-driven intravenous line. The patient experienced a significant fall in respiratory rate.

A slow continuous opioid infusion would only reflux slowly up an intravenous line if the cannula had become

obstructed. However, PCA has the potential for delivering several doses of opioid into the line over a short space of time. Hence I believe that antireflux valves should be mandatory in all situations when an opioid infusion is connected into a gravity fed intravenous infusion.

It may be that the use of such a system with an antireflux valve helps maintain the patency of intravenous cannulae by regular 'flushing'. However, I have no proof.

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More on automatic cardioverter-defibrillators

I wish to comment on the case report by Drs Carr and Whitley (*Anaesthesia* 1991; 46: 737-40) concerning the anaesthetic implications of the automatic implantable cardioverter-defibrillator (AICD). They clearly describe the working of the particular device in use at their hospital and their case report does illustrate some of the problems which the anaesthetist may encounter. I feel, however, that they have neglected to address the fundamental issues facing patients undergoing AICD implantation, such as choice of anaesthetic agents, the rationale for monitoring, and other problems such as that of external defibrillation. This is perhaps understandable as their impressions are based on limited clinical experience. I take issue with some of their statements, but at the same time take this opportunity to make further recommendations for this group of patients based on our experience. At our institution we carry out between two to four AICD implantations per week and to date, several hundred have been performed.

In contrast to the case reported by Drs Carr and Whitley, at our centre both implantation and testing of the device are performed under one general anaesthetic. They state 'full monitoring, including intra-arterial pressure and central venous pressure or pulmonary artery pressure, is mandatory'. Clearly an arterial line is essential as testing of the AICD involves the induction of ventricular fibrillation. Central venous access is essential for the passage of pacing electrodes and as a route for administering vaso-active drugs. Monitoring of central venous pressure is rarely helpful and the benefits of a pulmonary artery catheter must be weighed against the risk of inducing ventricular arrhythmias during catheter insertion [1].

Testing of the AICD involves inducing ventricular fibrillation and determination of the minimum energy required to revert to sinus rhythm. Therefore it would appear prudent to use anaesthetic agents which have minimal effects on cardiac conduction and defibrillation thresholds. If lignocaine is used for line placement, the amounts should be limited. For similar reasons, we do not use intravenous or topical tracheal lignocaine as a method of abolishing the stress response to laryngoscopy in these patients. For maintenance of anaesthesia a nitrous oxide, oxygen, narcotic technique is employed. If necessary, low concentrations of isoflurane can be added since, of the currently available inhalation agents, it probably has the least effect on cardiac conduction. As these patients are particularly prone to supraventricular arrhythmias in the postoperative period, the AICD is placed in the inactive mode for 2 to 3 days until the patient stabilises.

Drs Carr and Whitley correctly point out that external defibrillation may be required if the device fails, but do not elaborate on the potential difficulties which may arise. The energy requirement for defibrillation may be higher with application of paddles in the standard position if the current has to pass through the AICD patch electrodes. It has been recommended that external paddles be orientated at a right angle to a line joining the implanted patch electrodes to avoid this problem [2].

The AICD can be triggered by any form of electromagnetic radiation including electrocautery. If the latter is essential, it is best to follow the manufacturer's advice and place the AICD in the inactive mode. On a final note, we have been treating a patient with an implanted

AICD for myofascial pain at our pain control centre and would suggest that transcutaneous electrical nerve stimulation may be a relative contraindication. We advise our staff to wear gloves for the purpose of insulation when handling these patients as the AICD can discharge at any time.

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A reply

Thank you for the opportunity to comment on the letter from Dr Lee which raises several important points not discussed in our paper.

Our case report was an attempt to heighten awareness of the automatic implantable cardioverter-defibrillator, primarily amongst anaesthetists in the UK. I fully concede that our experience reflects that of the UK as a whole, which on this subject is still in its infancy compared to the United States. This was mentioned in our report and re-

emphasised by our references, which were almost all of United States origin.

Our paper was a case report and, as such, could not claim to be a fully exhaustive review article. Concerning the choice of anaesthetic agents used, with particular emphasis on their potential to induce supraventricular arrhythmias, we did make reference to induction agents in our first submission. This was later omitted because of space limitations. I accept that there is evidence to suggest an advantage in the use of isoflurane over enflurane in these patients, if a volatile agent is required [1]. I also fully accept and endorse his comments regarding the requirements of patient monitoring.

I thank Dr Lee for bringing to our attention the need for lignocaine limitation and for his comments relating how the problems of external defibrillation can be overcome in these patients. I am grateful for his advice regarding the avoidance of transcutaneous electrical nerve stimulation in these patients and for highlighting the need for all staff in contact with these patients to wear insulating gloves. The latter is not standard practice at Leeds, although they may change with experience.

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Reference

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Nil by mouth after midnight?

Patients who have elective surgery do find the experience stressful, as a recent survey demonstrated [1]. The issue concerning the timing of removal of dentures provoked much discussion, and our Department has subsequently changed its policy to allow denture wearers to keep their teeth in until after induction of anaesthesia.

However, one of the other major complaints relates to pre-anaesthetic fasting. This is usually related to the 'nil by mouth after midnight' rule, and the idea that anaesthesia cannot be induced without a minimum period of 4, or in some cases 6 h, of fluid restriction. Can anyone tell us where these figures come from? There is a considerable body of evidence to suggest that a small drink 2-3 h prior to surgery has no deleterious effect on either residual gastric volume or pH [2-4]. This also seems to hold true for patients who receive an opioid premedication [5]. Is there then any justification for the prolonged fluid deprivation that anaesthetists impose on elective surgery patients? We, as a Department, are giving consideration to a change of policy in this area and would be most interested to hear the opinions of other anaesthetists on the subject.

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Dental throat packs and airway protection

The interesting case described by Drs Whitely and Shaw (*Anaesthesia* 1992; **47**: 173) in which a throat pack failed to prevent an extracted tooth from passing into the nasopharynx, prompts me to raise the question of the purpose and use of such packs in oral and nasal surgery. Does their continuing widespread use in the intubated patient owe more to tradition than to any considered benefit? It is true that before the use of tracheal tubes and when such tubes were used uncuffed, a throat pack was

essential to prevent the aspiration of blood, secretions, teeth and dental fragments. However, if we are prepared to rely on the cuff of a tracheal tube to prevent the aspiration of potentially lethal stomach contents in obstetric and abdominal surgery, it would seem illogical not to rely upon it to prevent the aspiration of the relatively innocuous blood and secretions produced during nasal and dental surgery.

Deaths have certainly occurred due to the failure to

remove a throat pack following surgery and in spite of the most rigorous measures taken to prevent this disaster, it must remain a potential hazard. In oral surgery packs push the tongue forward, limiting the surgical field, restricting access, aggravating the operator's difficulties and increasing the likelihood of 'dropped tooth'. They do not reliably absorb blood or mucus and hence clearance of the pharynx, including the nasopharynx, by suction under direct vision is required before removal of the tube. Packs are, moreover, time consuming and messy to put in and,

even if vaginal tampons are used, they are traumatic to the pharyngeal mucosa and a cause of postoperative discomfort.

For all the above reasons, I stopped the routine use of throat packs some years ago and I have had no reason to regret this decision.

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Problem with the laryngeal mask airway

We wish to report a recent problem with an Intavent laryngeal mask airway (LMA). A 25-year-old ASA 1 female was scheduled to undergo an emergency anal stretch under general anaesthesia. After induction of anaesthesia with alfentanil and propofol, a size 3 LMA was inserted easily and the patient's lungs were readily ventilated via a Mapleson A system with 66% nitrous oxide in oxygen supplemented with halothane. She was transferred from the anaesthetic room to the operating theatre, where a large air leak from the breathing system was immediately apparent. Closer inspection showed that the LMA had broken transversely about halfway down the tube. The top half of the tube was pulled out, the cuff deflated, and the remainder of the mask removed by traction on the pilot tube. The airway was maintained for the rest of the procedure with a Guedel airway and facemask. The remainder of the anaesthetic was uneventful.

Examination of the laryngeal mask later showed that the tube had become very brittle and it was possible to break off easily small pieces. There was no discoloration of the tubing and routine inflation of the cuff prior to use had not revealed the problem with the tube. The manufacturer's instructions state that (a) the mask can be autoclaved up to 134°C, and should not be disinfected with glutaraldehyde [1]; (b) the LMA should be discarded if the tube is discoloured and (c) the LMA is not guaranteed for more than 10 uses ('while in practice it can usually be made to last much longer' [1]). The normal practice was that LMAs were washed in aqueous chlorhexidine immediately after use, and then sterilised using moist heat at 121°C in the CSSD. Occasionally, the masks were autoclaved in the operating department e.g. at a weekend, although even then the maximum temperature of 134°C was not exceeded. There are no data readily available regarding the effect of chlorhexidine on the LMA. No record is available of the

number of times this LMA had been used. We no longer use chlorhexidine to wash the masks but have not been able to devise an easy way to record the number of times the mask has been used, since use and cleaning of one mask may occur on many sites. After this incident we changed our policy of mask checking pre-operatively to include testing the flexibility of the tube by bending it, and recommended that this should be done routinely, since deterioration of the LMA may clearly occur in the absence of discoloration.

Colgate Medical have recently issued a warning of possible damage to LMAs. This is claimed to be due to over- or misuse, although checking of new LMAs is also recommended. We believe this example may be one of the cases prompting this. The testing regimen enclosed with the warning would probably have detected the damage in this case. These tests are more extensive than those in the instruction manual available in our hospital at that time [1], although the checklist is incorporated in the second edition of this manual [2] (this new regimen also includes our above recommendation of bending the LMA, although the apparent intent is to detect kinking rather than brittleness). We support the general adoption and publicising of this testing regime.

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A. SPRINGER
J. HINDMARSH

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Use of 15 mm tubing with the Humphrey ADE breathing system

I read with interest the report by Murphy and Rabey (*Anaesthesia* 1991; 46: 1000) regarding the malfunction of the Penlon IPD ventilator disconnection alarm when using 15 mm tubing with the Humphrey ADE system. I have become increasingly aware that there are many different 15 mm tubes available and that each have their own gas flow characteristics. These appear to be particularly related to the corrugations of the internal bore. High resistance to flow could affect ventilator and alarm functions.

All my published work on the Humphrey ADE system has been conducted using smooth bore tubing (Anaquip UK, Worthen, Shrewsbury, Shropshire) which has a relatively low resistance to flow, as flow through it is laminar; its diameter is more or less constant along the length of the tube. Because I found that this tubing had resistance values less than the larger tubing used with the

Bain or Lack systems, I recommended its use for adults. However, I have recently evaluated the 15 mm tubing supplied by Intersurgical (Twickenham, Middx), as I had noticed that it was considerably more difficult to breathe through it. Turbulence appeared to occur with larger breaths, as a whistle could be heard from within the tube, the turbulence probably being caused by the internal corrugations.

As a result of these latter subjective observations and the many requests for the flow characteristics of the 15 mm tubes I have recommended, I have compared the flow characteristics along the following commonly used tubing: smooth bore 15 mm tube supplied by Anaquip UK, corrugated 15 mm tube from Intersurgical, coaxial Bain system tubing and coaxial Lack tubing. The pressure drop across the two ends of each tube were measured with a

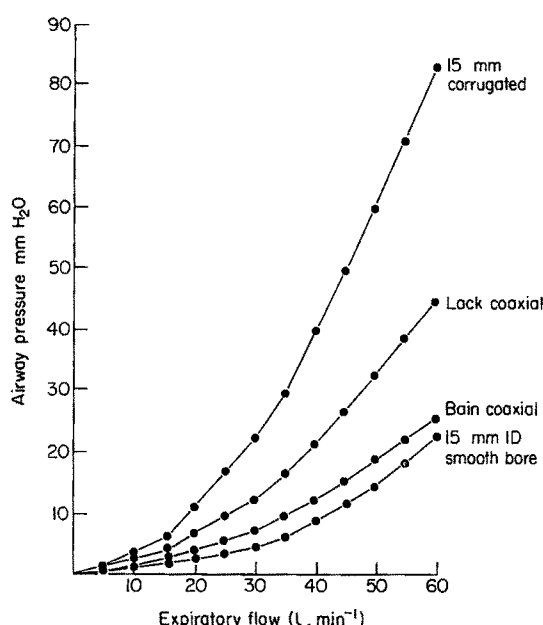


Fig. 1. Flow characteristics of various commonly used tubing for anaesthetic breathing systems as measured by the use of a simple water manometer. 15 mm smooth bore tubing is recommended for universal use.

water manometer with varying flows of oxygen being introduced at one end. The tubes were all 1.5 m in length. The results are in the Figure.

At low flows, the differences between the various tubes

were not of great clinical significance, but at higher flows the differences were marked. At all flows the 15 mm smooth bore tubing offered the least resistance. The Bain tubing offered a little more resistance, but significantly less than the Lack. The corrugated 15 mm tubing offered significantly more resistance than any of the other tubes, confirming the subjective observations.

The possibility of high airway pressure is thus greatest with corrugated tubing. Thus at a flow of 45 l.min^{-1} , which Murphy and Rabey were using with the ADE system with the Penlon ventilator (equivalent a setting of 750 ml.s^{-1}), the resistance to flow through the corrugated tubing would be $68 \text{ mmH}_2\text{O}$. Murphy and Rabey do not state what type of tubing was being used, but the different flow characteristics of various tubing may explain the failure of the IDP alarm. It is thus possible that if corrugated 15 mm tubing was used, the extra resistance, when added to the total flow resistance through a Penlon ventilator, could cause an IPD ventilator alarm failure. When using smooth bore tubing I could not reproduce the problem described by Murphy and Rabey.

In conclusion, only smooth bore 15 mm tubing can be recommended as acceptable as a universal tubing for the ADE system. As resistance is directly proportional to length, the use of two lengths (3 m) is acceptable, provided the function of any disconnect alarm is checked. The use of corrugated 15 mm is unsuitable for adults, though it remains suitable for children.

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Eliminating pollution in paediatric and dental anaesthesia

I read with interest the suggestion by Kumar (*Anaesthesia* 1991; 46: 792) regarding yet another device to reduce operating theatre pollution with Ayre's T-piece. I offer a suggestion that could totally eliminate the problem of pollution in both paediatric and dental anaesthesia.

The prevention of pollution can be tackled in two ways; reduce the source and capture the waste. Most devices try to capture the waste, but tend to be clumsy or limiting (Kumar warns that assisted ventilation is not possible with his suggestion). The other alternative is to reduce the fresh gas flow. However, the most recognised breathing system for children is Ayre's T-piece which, for spontaneous respiration, is known to be relatively inefficient as it has the characteristics of a Mapleson E system. Many use 6 l.min^{-1} as a standard flow.

I would like to suggest that both methods of pollution control can be safely achieved with the use of the Humphrey ADE breathing system. Fresh gas flows can be significantly reduced while expiratory gases are contained within the system. A recent report [1] recommended the use of the Humphrey ADE breathing system for children. The authors compared it to the T-piece and concluded that 'in contrast, the Humphrey ADE system is extremely efficient during spontaneous respiration in the A mode allowing a fresh gas flow of 3 l.min^{-1} to be used with a considerable margin of safety in children under 25 kg ... Scavenging from the ADE system is simple, as expired gases are led back to the manifold.' At the anaesthetic machine end, the waste gases are scavenged by any of the numerous devices available (I have found that described by Enderby *et al.* [2] to be effective, simple and safe). It should be noted that scavenging does not compromise the simplicity of the system or the lightness of the tubing. Spontaneous, manual

or controlled ventilation can be instituted at any time, without alteration to the system and without compromising pollution control.

The same principle can be applied to pollution control during dental anaesthesia using the McKesson mask with the ADE system. The ADE system is used in its efficient 'A' mode for spontaneous respiration, while expired gases are collected and returned to the ADE manifold (Fig. 1). The system is set up as follows: fresh gas is supplied from the ADE manifold through a 1.5 m long 15 mm ID smooth bore tube to the dental mask. The valve on the mask is never used so it can be closed or completely removed and blocked off. When the patient breaths out, expired gases pass out of the mask opposite the fresh gas inlet into the expiratory limb (also a 15 mm smooth bore tube) which conducts waste gases back to the ADE manifold on the anaesthetic machine. Expired gases are scavenged at this point.

In conclusion, operating theatre pollution can be controlled by minimising gas flows and ensuring the

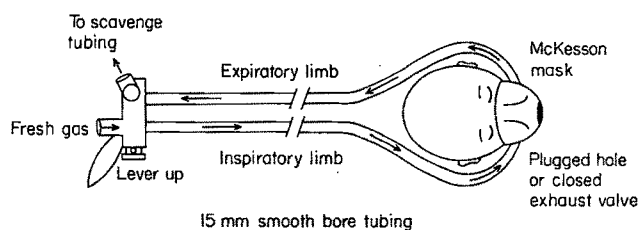


Fig. 1. The set up for using the Humphrey ADE system with the McKesson dental mask. Low flows and scavenging reduce pollution to a minimum.

complete capture of waste gases. The Humphrey ADE system achieves both.

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References

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Obituaries

Milledge, Betty Avril, MB, ChB, DA. Formerly Clinical Assistant in Anaesthesia, Northwick Park Hospital, Harrow, Middlesex. Qualified from University of Birmingham in 1953.

Hutchinson, J.R.S., MB, ChB, FFARCS. Formerly Consultant Anaesthetist, Victoria Infirmary, Glasgow. Qualified from Glasgow University in 1946.

International congress calendar

1992

- 2-3 July.** Bordeaux, France. *Tracheal Intubation*.
Information: Anne-Marie CROS, Department of Anesthésie Réanimation, Hôpital Pellegrin, Place Amélie Raba-leon 33076, Bordeaux Cedex, France.
- 12-16 August.** Dunedin, New Zealand. *Intravenous and Inhalational Anesthesia*.
Information: Organizing Secretariat CANZ 1991, Department of Anaesthesia and Intensive Care, Dunedin Hospital, Dunedin, New Zealand.
- 19-21 August.** Edinburgh. *VIIIth Edinburgh Anaesthesia Festival*.
Information: Edinburgh Anaesthesia Festival, University Department of Anaesthetics, Royal Infirmary, Lauriston Place, Edinburgh EH2 9YW.
- 25-26 August.** Kiekrz, Poznan. *1st Refresher Course of the European Academy of Anaesthesiology*.
Information: Prof. dr med. Laura Wolowicka, 1st Refresher Course EAA, Klinika Intensywnej Opieki Medycznej, 60-479, Poznan (Poland), ul. Lutycka.
- 27-30 August.** Poznan, Poland. *European Academy of Anaesthesiology (Open) Refresher Course and (Members only) Scientific Meeting*.
Information: Professor M.D. Vickers, Department of Anaesthesia, University of Wales College of Medicine, Heath Park, Cardiff CF4 4XN.
- 2-4 September.** Southampton. *Ultrasound Angiography 92. 2nd International Conference*.
Information: Conference Secretariat, Ultrasound Angiography 92, P.O. Box 15, Eastleigh, Hampshire SO5 5X9.
- 9-11 September.** Bournemouth. *Linkman and Annual Scientific Meeting*.
Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA.
- 14-16 September.** Leicester. *British Sleep Society, 4th Annual Meeting, University of Leicester*.
Information: Dr C.D. Hanning, Sleep Disorders Clinic, Leicester General Hospital, Leicester LE5 4PW.
- 14-18 September.** Jerusalem, Israel. *5th International Congress, The Pain Clinic*.
Information: Secretariat, The Pain Clinic, P.O. Box 50006, 61500 Tel Aviv, Israel.
- 17-20 September.** Halkidiki, Macedonia, Greece. *2nd Congress of Anaesthesiology and Intensive Medicine*.
Information: Secretariat, 2nd Congress of Anaesthesiology and Intensive Medicine, P.O. Box 10058, 54101 Thessaloniki, Greece.
- 22-25 September.** Cuba. *First International Symposium on Brain Death*.
Information: Dr Calixto Machado, President of the Organizing Committee, Zona Postal 4, Apartado 4268 Ciudad de La Habana 10400 Cuba.
- 1-2 October.** London. *Continuing Medical Education and Training in Europe: the future*.
Information: Dr W.M.N. Nicholls or Mrs J.M. Coops, The Conference Office, c/o The Fellowship of Postgraduate Medicine, 6 St Andrew's Place, London NW1 4LB.

- 8-10 October.** Mainz, Germany. *13th International Congress on Computers in Critical Care and Pulmonary Medicine*.
Information: Professor W. Dick, Klinik für Anästhesiologie, Langenbeckstrasse 1, Postfach 3960, 6500 Mainz, Germany.
- 17-20 October.** New Orleans. *American Society of Anesthesiologists Annual Meeting*.
Information: Executive Secretary, ASA, 515 Busse Highway, Park Ridge IL 60068, USA.
- 4-6 November.** Israel. *The Sixth International Symposium on Anesthesia and Intensive Care*.
Information: Dr A. Fisher, Division of Anesthesiology, Soroka Medical Center, Beer-Sheva 84101, Israel.
- 20-21 November.** Brighton, England. *1st CPR Congress of the European Resuscitation Council*.
Information: Dr D.A. Zideman, Department of Anaesthesia and Critical Care, Hammersmith Hospital, Du Cane Road, London W12 0HS.
- 12-16 December.** New York. *46th Postgraduate Assembly in Anesthesiology*.
Information: Kurt G. Becker, Executive Director, The New York State Society of Anesthesiologists, Inc., 411 East 42nd Street, Suite 1605, New York 10017, USA.

1993

- 16-23 January.** Cancun, Mexico. *11th Annual Symposium, Clinical Update in Anesthesiology*.
Information: George Silvay, WACTVA, 1408 Madison Avenue, Suite 2C, New York, NY 10029, USA.
- 22-23 January.** Leuven, Belgium. *Anesthetic Mishaps*.
Information: Prof Dr H. Van Akenh, Department of Anesthesiology, University Hospitals K.U. Leuven, Herestraat 49, B-3000 Leuven, Belgium.
- 30 January-6 February.** Aspen, Colorado. *New Horizons in Anesthesiology*.
Information: Miss Kathleen A. Maitland, Department of Anesthesiology, Emory University at Crawford Long Hospital, 25 Prescott Street, 5404 Glenn, Atlanta GA 30308, USA.
- 12-16 February.** Utah. *38th Annual Postgraduate Course in Anesthesiology—'Anesthesiology: Today and Tomorrow'*.
Information: Vicky Larson, Department of Anesthesiology, University of Utah School of Medicine, 50 North Medical Drive, Salt Lake City, Utah 84132, USA.
- 12-15 March.** Hong Kong. *7th Congress of the Western Pacific Association of Critical Care Medicine*.
Information: Secretariat, International Conference Consultants Ltd, Suites A & B, 16th Floor Casey Building, 38 Lok Ku Road, Sheung Wan, Hong Kong.
- 14-18 March.** Cape Town, South Africa. *South African Society of Anaesthetists Jubilee Congress*.
Information: Mrs S. Elliot, Postgraduate Education Centre, UCT Medical School, Observatory 7925, Cape Town, South Africa.

19-20 March. Brussels. *Second European Congress on Ambulatory Surgery.*

Information: CI De Lathouwer, M.D. Brussels One Day Clinic, avenue du Duc Jean 71-73, B-1080 Brussels, Belgium.

19-23 March. San Diego, California. *67th Congress of the International Anesthesia Research Society.*

Information: Anne F. Maggiore, Executive Director, International Anesthesia Research Society, 2 Summit Park Drive, Suite 140 Cleveland, Ohio 44131, USA.

21-23 April. Sheffield. *Anaesthetists in Training Annual Scientific Meeting and Linkman Conference, Octagon Centre, Sheffield.*

Information: The Honorary Secretary of the Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA.

29 April-2 May. North Carolina. *Meeting of the Association of University Anesthetists.*

Information: Francis M. James III, Department of Anesthesia, Wake Forest University Medical Center, 300 S Hawthorne Road, Winston-Salem, North Carolina 27193, USA.

13-16 May. Seattle, WA, USA. *American Society of Regional Anesthesia, 18th Annual Meeting, Refresher Course, Workshops.*

Information: ASRA, P.O. Box 11086, Richmond, VA 23230-1086, USA.

10-13 June. Boca Raton, Florida. *Annual Meeting of the Florida Society of Anesthesiologists.*

Information: Florida Society of Anesthesiologists, 6501 25 Way S. Ste D, St Petersburg, FL 33712, USA.

1-4 September. Liverpool. *European Course and Congress in Paediatric Anaesthesia.*

Information: Dr P.D. Booker, Alder Hey Hospital, Liverpool L12 2AP.

21-24 September. Ghent. *Update in Cardiac Surgery, Anesthesia and Intensive Care. 2nd Meeting.*

Information: Eric Mortier, University Hospital Ghent, De Pintelaan 185 B-9000 Ghent, Belgium.

22-24 September. Glasgow. *Linkman Conference and Annual Scientific Meeting. Joint Meeting between the Association of Anaesthetists of Great Britain and Ireland, and the Canadian Society of Anaesthetists.*

Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA.

9-13 October. Washington DC. *American Society of Anesthesiologists Annual Meeting.*

Information: Executive Secretary, ASA 515 Busse Highway, Park Ridge, IL 60068 USA.

1994

7-10 April. Chicago. *American Society of Regional Anesthesia 19th Annual Meeting, Refresher Course, Workshops.*

Information: ASRA P.O. Box 11086, Richmond, VA 23230-1086, USA.

24-27 April. Montreal, Canada. *16th Annual Meeting and Workshops Society of Cardiovascular Anesthesiologists.*

Information: SCA, P.O. Box 11086, Richmond, VA 23230-1086, USA.

7-9 September. Brighton. *Linkman Conference and Annual Scientific Meeting.*

Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA.

2-7 October. Jerusalem. *European Congress of Anaesthesiology.*

Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA.

1995

7-10 May. Pennsylvania. *17th Annual Meeting and Workshops Society of Cardiovascular Anesthesiologists, Philadelphia, Pennsylvania, USA.*

Information: SCA P.O. Box 11086, Richmond, VA 23230-1086, USA.

13-15 September. Birmingham. *Linkman Conference and Annual Scientific Meeting.*

Information: The Honorary Secretary, Association of Anaesthetists of Great Britain and Ireland, 9 Bedford Square, London WC1B 3RA.

1996

5-8 May. Utah, USA. *18th Annual Meeting and Workshops Society of Cardiovascular Anesthesiologists. Salt Lake City.*

Information: SCA P.O. Box 11086, Richmond, VA 23230-1086, USA.

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SOTER NA, WASSERMAN SI, AUSTEN KF. Cold urticaria: release into the circulation of histamine and eosinophil chemotactic factor of anaphylaxis during cold challenge. *New England Journal of Medicine* 1976; **294**: 687–90.

Corporate author

The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology. Recommended method for the determination of gamma-glutamyltransferase in blood. *Scandinavian Journal of Clinical Laboratory Investigation* 1976; **36**: 119–25.

Anonymous. Epidemiology for primary health care. *International Journal of Epidemiology* 1976; **5**: 224–5.

BOOKS AND OTHER MONOGRAPHS

Personal author(s)

OSLER AG. Complement: mechanisms and functions. New York: Prentice-Hall, 1976.

Corporate authors

American Medical Association Department of Drugs. *AMA drug evaluations*, 3rd edn. New York: Publishing Sciences Group, 1977.

Editor, compiler, chairman as author

RHODES AJ, VAN ROOVEN CE, comps. *Textbook of virology: for students and practitioners of medicine and other health sciences*, 5th edn. Baltimore: The Williams & Wilkins Co., 1968.

Chapter in book

WEINSTEIN L, SWARTZ MN. Pathogenic properties of invading micro-organisms. In: SODEMAN WA Jr, SODEMAN WA, eds. *Pathologic physiology: mechanisms of disease*. Philadelphia: W.B. Saunders, 1974: 457–72.

Agency publication

National Center for Health Statistics. Acute conditions: incidence and associated disability, United States, July 1968–June 1969. Rockville, MD: National Center for Health Statistics, 1972. (*Vital and health statistics, Series 10*: Data from the National Health Survey. No. 69) [DHEW publication No. (HSM) 72-1036].

OTHER ARTICLES

Newspaper article

SHAFFER RA. Advances in chemistry are starting to unlock mysteries of the brain: discoveries could help to cure alcoholism and insomnia, explain mental illness. How the messengers work. *Wall Street Journal* 1977 Aug 12: 1(col 1), 10(col 1).

Magazine article

ROUCHE B. Annals of medicine: the Santa Claus culture. *The New Yorker* 1971 Sept 4: 66–81.

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REVIEW JOURNALS

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Anaesthesia

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Volume 47 Number 8 August 1992

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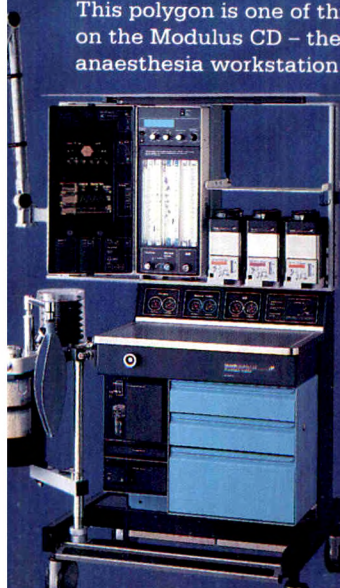
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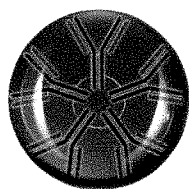
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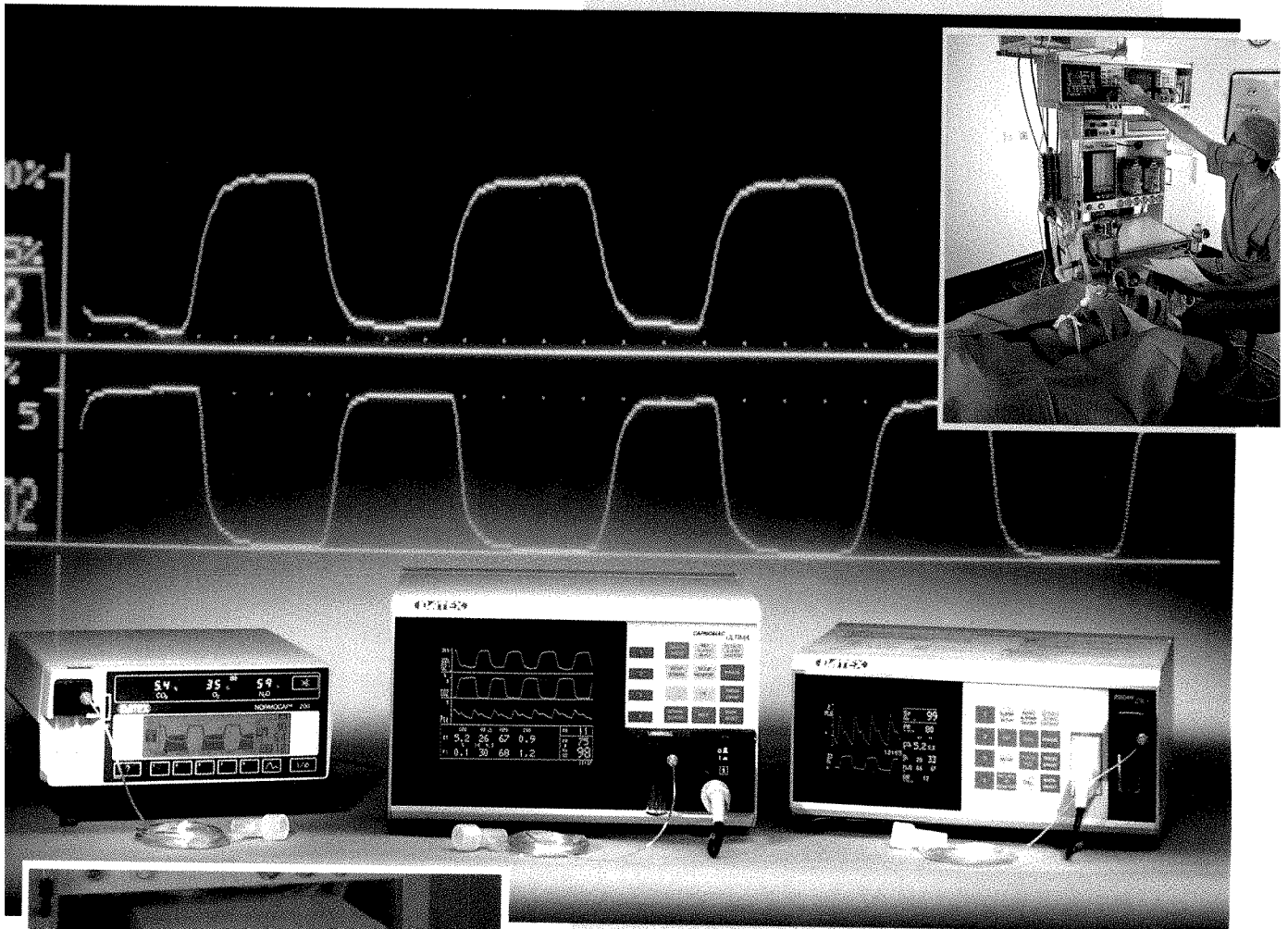
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Editorial

Anaesthetists, nurses, and pre-operative visiting

The pre-operative visit of patients by their anaesthetist is considered by both the Royal College of Anaesthetists and the Association of Anaesthetists to be an essential part of modern anaesthetic management and good practice [1]. The Association *Survey of anaesthetic practice* [2] showed that the majority of patients do indeed receive a pre-operative visit from the anaesthetist responsible for their clinical care.

Is a pre-operative visit important, and if so for whom, and by whom should it be performed? The question is particularly relevant at a time when management, concerned to maximise the throughput of scheduled cases through an operating theatre, might be tempted to regard time allocated for ward work as wasted time, and when operating theatre nurses, anxious to preserve professional status in the wake of the Bevan report [3], attempt to establish their own system of pre-operative visiting.

A pre-operative visit has been shown to be consistently beneficial for patients. Fifteen years ago, Leigh *et al.* [4] claimed that a representative of the anaesthetist, possibly a nurse, might be usefully employed in pre-operative visiting. In 1991, however, the observations of Lonsdale and Hutchinson [5] that patients expect to 'assess the person to whom they are entrusting themselves' has to be taken seriously. In today's medicolegal climate where the right of a patient to be informed is stressed, it is a brave anaesthetist who is prepared to delegate the task of visiting to anyone other than a colleague (e.g. a trainee anaesthetist who will be also involved in the clinical care). Given that anaesthetists should make a strong case for contracts that reflect pre-operative commitments, what are we to make of the trend towards operating theatre nurses making a pre-operative visit?

There is nothing new in this idea, as Leigh and his colleagues [4] showed. What is new is the concept that operating theatre nursing care can be planned in the same way as it is on the wards, on an individual basis, with a nurse being responsible for the patient's well-being throughout the peri-operative period [6,7]. This is a concept that is at odds with the obligations of the anaesthetist, who is directly and personally responsible for all aspects of the well-being of the anaesthetised patient.

An anaesthetist who is involved in discussion with nursing colleagues about operating theatre nurse visiting should be aware that what is being discussed is not the sort of visit that Leigh *et al.* recommended. Rather it is a separate evaluation of an autonomous profession to gather information about nursing requirements and to provide general reassurance. While we would not wish to interfere with the way nurses work, it must be remembered that there is a potential for conflict if the visits of nurse and doctor take place without mutual discussion. For instance, the reassurance of a nervous patient that modern anaesthesia involves 'no mask, just a little needle' may be inappropriate for the patient with a full stomach or a hiatus hernia who is required to breathe oxygen before induction. Counselling on the possibility

of complications of anaesthesia is similarly the responsibility of the anaesthetist, although operating theatre nurses may be asked about these matters.

Anaesthetists should, of course, recognise that patients may confide in nurses to an extent that they do not in doctors, and be prepared to discuss individual problems with ward nurses, whom they may also advise about appropriate patient reassurance. It is not necessary for a theatre nurse to be involved at this stage. As one nurse teacher puts it [8]; 'a bright smile and a grilling from a theatre nurse ... visiting for a few brief minutes are not going to help. Helping the patient with pre-operative anxiety is more properly the province of the ward nurse'.

The visiting of patients by operating theatre nurses may be educational and good for nurses' morale and professional status. However, in a service in which the professional's time is a valuable resource, a manager could see the visit of operating theatre nurse and anaesthetist as duplication. Under these circumstances it is vital that anaesthetists leave management in no doubt that pre-operative visiting by anaesthetists is essential for safe practice and that time is allocated in contracts for this function. This duty should be assiduously carried out; failure to visit a patient pre-operatively should occur only exceptionally. This is a duty which cannot be delegated to nurses, and properly carried out by the anaesthetist should make an operating theatre nurse's visit unnecessary.

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Low dose amitriptyline in the treatment of chronic pain

H. J. MCQUAY, D. CARROLL AND C. J. GLYNN

Summary

The analgesic efficacy of amitriptyline 25 mg was compared with placebo in 41 patients with chronic (more than 3 months) nonmalignant pain, using a double-blind randomised multiple-dose 3-week treatment period crossover design. Amitriptyline 25 mg provided significantly greater efficacy than placebo, with significant differences evident within the first week. There was no significant difference on mood scores between amitriptyline and placebo. The results suggest that surprisingly low doses of amitriptyline may be effective without substantial adverse effects, that the effect is evident early, and that the effect is distinguishable from any effect of the amitriptyline on mood.

Key words

Pain; chronic.

Ataractics; antidepressants, tricyclic, amitriptyline.

The precise role of antidepressants as unconventional analgesics in pain management is uncertain. It is believed that antidepressants are effective in relieving some symptoms, particularly burning or aching pain, in pain syndromes where the nervous system has been damaged. Controlled studies in syndromes such as diabetic neuropathy [1–4], postherpetic neuralgia [5–8] and facial pain [9, 10] support this belief. Antidepressants, however, are used empirically in a wide range of other pain syndromes with apparent clinical benefit. It is unclear how valid the extrapolation is from controlled studies within a particular syndrome to patients who are less easily categorised. Antidepressant treatment has been shown to be superior to placebo in patients with heterogeneous pain syndromes [11, 12], although negative results have also been reported [13].

How antidepressants work as analgesics is also a mystery. Tricyclic antidepressants block α_1 adrenergic, muscarinic and H_1 -histamine receptors, and also block noradrenaline and serotonin re-uptake. Two of the questions which remain unanswered are which of these properties are the relevant ones, and which are the site(s) of action, the brain or the spinal cord, direct or indirect via descending influence. The choice of the 'correct' dose is also a problem. The dose–response curve for analgesia in the chronic pain setting appears to be shifted to the left of the dose–response for depression [14], and the significant analgesic effect achieved with low doses supports this conclusion [12]. The aim of this study was to show whether or not low-dose antidepressants had an effect in nonspecific

chronic pain, because the literature is unclear and because a positive finding would justify empirical clinical practice.

Methods

Study design

The study was a double-blind randomised multiple-dose crossover comparison of amitriptyline 25 mg with placebo. Ethics committee approval was obtained, and 41 outpatients attending the Oxford Regional Pain Relief Unit with chronic nonmalignant pain of more than 3 months' duration took part. Written and verbal explanation were given to the patients before starting the treatment. The study lasted 6 weeks, with two treatment periods of 3 weeks each and no washout period.

Patients

Adult patients of either sex were eligible for the study. They had to be capable of reliable communication with the observer, and to be free from clinically overt cardiac, renal or hepatic disease. Patients were permitted to take their regular conventional analgesic drugs at constant dosage during the study. They were at home, and only returned to hospital for the clinical assessments. The study was designed to compare amitriptyline and placebo in patients who would have been prescribed amitriptyline as an analgesic on clinical empirical grounds. These grounds were

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that the pain had previously not responded well to conventional analgesics, or that the character of the pain, burning or aching, was predictive of such response. Twenty-five mg amitriptyline is our usual starting dose in such circumstances.

Treatment and diaries

The treatments were presented as identical two-tone grey capsules, containing either sustained release amitriptyline 25 mg (Lentizol SR, Parke-Davis, Eastleigh) or identical matching placebo capsules. The order of the treatments was randomised; capsules were identifiable only by patient and treatment number. All clinic assessments were made by one nurse observer (D.C.), who was responsible for the patients during the study period. The patients filled in diaries daily with the 'Oxford Pain Chart' [15] to record the following information for the 6 weeks of the study:

Patient diary assessments

(1) Daily pain intensity was measured by a 4-point categorical scale (none = 0, mild = 1, moderate = 2, severe = 3). The patients were asked 'How bad has your pain been today?' (2) Daily pain relief was recorded using a 5-point categorical scale (none = 0, slight = 1, moderate = 2, good = 3, complete relief = 4). The patients were asked 'How much pain relief have the capsules given you today?' (3) Daily adverse effect incidence was recorded. The patients were asked 'Has the treatment upset you in any way?' (4) A weekly global-rating score was completed (poor = 0, fair = 1, good = 2, very good = 3, excellent = 4). The patients were asked 'How effective was the treatment this week?'

Clinic assessments

Initial clinic visit. Demographic details were recorded with a medical history and details of pain and current drug therapy. Patients were asked about their current pain intensity and typical pain intensity over the previous week (none = 0, mild = 1, moderate = 2 and severe = 3). A visual analogue scale was used to measure pain intensity, using a 100 mm line marked 'least possible pain' at one end and 'worst possible pain' at the other end. Mood was measured with a 100 mm line marked 'worst I could feel' at one end and 'best I could feel' at the other. An eight-word randomised scale for pain intensity [16] ('no pain' = 0 to 'excruciating' = 7, in that order), the McGill Pain Questionnaire [17] and a weekly retrospective sleep scale (poor = 0, fair = 1, good = 2, very good = 3, excellent = 4) were also used. The patients were asked to continue any analgesic drugs at constant dose. They were asked to take one capsule of the study medication at night 1 h before going to bed, and to fill in the diary at that time. They then went home with the diary, which they were asked to complete daily for 3 weeks.

Clinic visits. Patients attended the clinic after both 3-week treatment periods. Diaries were collected. Pain intensity visual analogue was recorded as for the initial visit, together with a visual analogue for pain relief, using a 100 mm line (marked 'no relief of pain' at one end and 'complete relief of pain' at the other). The McGill Pain Questionnaire was repeated, together with a five-point

categorical rating scale for pain relief, four-point scales for current pain intensity and typical pain intensity during the previous week, mood and sleep. An overall global score (poor = 0, fair = 1, good = 2, very good = 3, excellent = 4) was made for the treatment and details of any adverse effects were recorded, as well as any comments. If the patient withdrew before a 3-week treatment period had elapsed the measurements were made at the time of withdrawal, and the patient went on to the second arm of the crossover as appropriate. Patients returned any remaining tablets at the clinic visit, and these were counted as a compliance check.

At the final clinic visit (end of second 3-week treatment period), the patients were asked to give a preference for either the first or the second treatment. The individual patient code was then broken to allow for further appropriate treatment: satisfactory relief, continue; relief with adverse effects, decrease dose; no relief and no adverse effects, increase dose.

After the study, when patients came back to the clinic for routine follow-up, the nurse observer obtained details about continued amitriptyline use, recording response, dosage and adverse effects.

Analysis

McGill scores for words chosen and sum rank were made. From the daily diary pain intensity and pain relief recordings, mean pain intensity and pain relief were calculated for each week of treatment. Incomplete daily recordings were omitted from analysis. This calculated data, and the other analgesic and mood score raw data, were compared within each patient for the two treatments using the Wilcoxon signed-ranks matched-pairs test. Chi-square with Yates' correction was used to compare adverse effect incidence for the two treatments. Order effects were checked by comparing scores within treatments across periods using analysis of variance. Results are presented as mean (SEM). Significance was assumed at the $p < 0.05$ level.

Results

Forty-one patients were recruited of whom eight withdrew, leaving 33 to be used for the analysis (Fig. 1). The reasons for withdrawal were patient decision to withdraw (1 patient after 0 doses, 1 after 1 and 1 after 2), noncompliance (4 neither took medication nor completed diary) and severe adverse effects (1 patient vomited after 1 dose of amitriptyline). Details of the 33 patients who completed the study are given in Table 1. All these 33 patients completed the first week of both treatments. Four patients subsequently dropped out of the amitriptyline arm; two (numbers 1 and 37), at the end of the first week and two, (numbers 27 and 40), at the end of the second week. Six patients dropped out of the placebo arm; two, (numbers 1 and 27), at the end of the first week, one, (number 21), during the second week and three, (numbers 7, 19 and 29), at the end of the second week. These patients went on to receive the second treatment in the same way as the first.

Analgesic measures

The results of the clinical assessments and diary measures are shown in Table 2. Significantly greater efficacy was

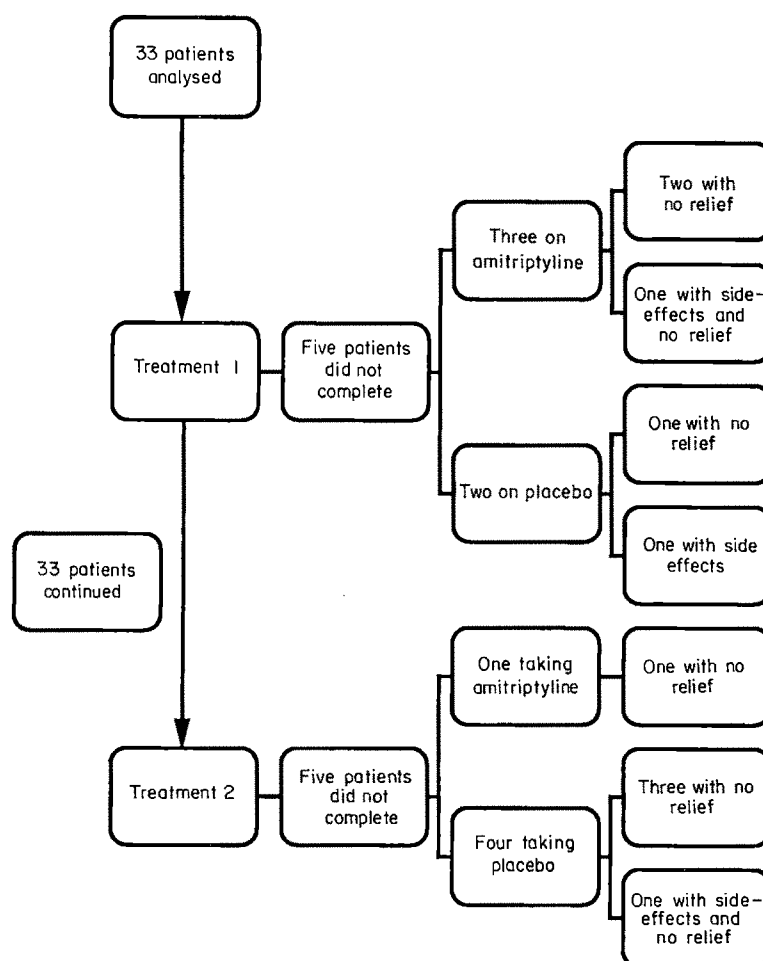


Fig. 1. Trial outcome for each treatment.

found for amitriptyline compared with placebo on the weekly global scores, mean pain intensity score for week 2 and mean pain relief scores for weeks 2 and 3. The mean daily pain relief scores for amitriptyline and placebo are shown in Fig. 2. Amitriptyline produced significantly greater pain relief within the first week (Fig. 2), and the mean global score for the first week was significantly higher

with amitriptyline (Table 2). When the daily pain relief scores were compared for the 21 days of each treatment, in 14 patients the scores were significantly higher with amitriptyline than with placebo (Table 3).

The clinic assessments showed significantly lower current pain intensity, McGill Total Score and visual analogue pain intensity, and significantly higher pain relief (visual

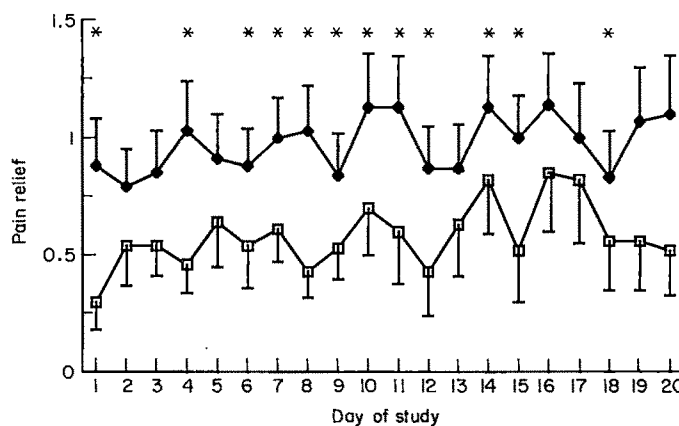


Fig. 2. Mean (SEM) daily pain relief scores by day of study for —●— amitriptyline 25 mg and —□— placebo. *denotes significant difference using Wilcoxon signed-ranks matched-pairs test.

Table 1. Patient details.

No.	Sex	Age; years	Height; cm	Weight; kg	Diagnosis	Site of pain	Duration; months	Character of pain
1	F	83	150	55	cervical spondylosis	back of head radiating to front	12	crawling
3	M	38	173	73	back pain	lumbar spine, both buttocks	12	throbbing ache
4	F	61	158	76	back trauma	lumbar and thoracic region	144	spasms like labour
5	M	69	179	89	post-herpetic neuralgia	C ₃	3	stabbing, sensitive
6	F	69	158	67	facet joint	R iliac fossa and groin	96	tight, inflamed feeling
7	F	73	165	83	multiple pains	abdomen/back/neck/chest/head	120	shooting and aching
8	M	34	173	65	back pain	lumbar spine and left leg	120	dull throbbing
9	M	33	175	104	back trauma	back/anterior aspect legs, knees	27	pulling
10	F	68	158	75	autonomic neuralgia	R foot radiating up leg	168	tingling, shooting
12	M	46	185	89	back pain	low back radiating to L leg	72	numb, tight
15	F	51	163	51	abdominal pain	abdomen, R back and groin	36	burning
16	F	74	155	57	Raynaud's/scleroderma	skull/R side face and head	8	constant ache and stabbing
17	F	45	152	43	post subarachnoid	headache radiating back to front	30	burning, squeezing
18	F	75	152	57	facial pain	L head/face/neck/shoulders	16	itch, dull ache
19	M	55	173	86	postsurgical chest pain	sternum/chest pain T _{8,9,10}	33	tight, bursting, shooting
20	F	55	158	54	facial pain	neck, shoulders, head	120	spasms, shooting
21	F	60	175	108	back pain	back and legs	12	aching
24	F	48	163	127	diabetic neuropathy	shoulder, hand and wrist	66	numb, tingling
26	F	74	156	76	coccydynia	coccyx and both legs	348	numb, weak
27	F	57	160	60	back pain	back, neck, head	136	throbbing
28	M	60	179	81	postsurgical chest pain	L chest	24	tight, stabbing
29	F	80	155	51	facial pain	face and neck	120	shooting
30	M	43	185	67	back pain	back, hips, R leg	84	tingling
31	F	41	165	64	1° lymphoedema	L leg	36	heavy, shooting
32	F	52	178	80	2° lymphoedema	leg	36	dull
33	F	54	163	108	2° lymphoedema	arm	24	aching, shooting
34	F	66	152	64	coccydynia	coccyx, low back, groin, legs	24	tight, gnawing
35	M	48	185	95	back pain	back, calf (L) and buttock	14	cramping, pins and needles
36	F	27	160	59	trauma	knees	192	aching, burning, shooting
37	M	76	178	67	back pain	L ₅ , S ₁	240	tender
38	F	55	155	52	post-herpetic neuralgia	buttocks, girdle	5	deep aching
39	F	54	152	66	multiple pain	multiple sites	48	burning, tingling
40	M	60	175	89	post stroke pain	hemibody	39	numb
Mean		57	166	74		Median	36	
sem		3	2	3		range	3-148	

Table 2. Analgesic and mood scores. Values are expressed as mean (SEM).

Baseline	<i>n</i>			
Current pain intensity	2.1 (0.2)	33		
Typical pain intensity	2.6 (0.1)	33		
McGill no. of words chosen	11.7 (0.8)	33		
McGill total score	29.8 (2.2)	33		
VAS pain intensity	68.0 (3.9)	33		
Eight word pain intensity scale	5.4 (0.2)	33		
Mood	39.9 (44.4)	33		
Sleep	1.1 (0.3)	15		
During treatment	<i>n</i>			
Global week 1	0.3 (0.6)	33	0.7 (0.1) 33	* <i>p</i> < 0.01
Global week 2	0.4 (0.1)	30	0.7 (0.1) 31	* <i>p</i> < 0.001
Global week 3	0.5 (0.2)	27	0.8 (0.2) 29	* <i>p</i> < 0.01
Mean of daily diary entries	<i>n</i>			
Pain intensity week 1	2.2 (0.1)	33	2.2 (0.1) 33	
Pain intensity week 2	2.2 (0.1)	30	2.1 (0.1) 31	* <i>p</i> < 0.02
Pain intensity week 3	2.1 (0.1)	29	2.1 (0.1) 30	
Pain relief week 1	0.8 (0.1)	33	1.1 (0.2) 33	
Pain relief week 2	0.6 (0.2)	30	1.0 (0.2) 31	* <i>p</i> < 0.001
Pain relief week 3	0.6 (0.2)	29	1.0 (0.2) 30	* <i>p</i> < 0.001
After treatment (clinic assessment outcome measures)	<i>n</i>			
Current pain intensity	2.4 (0.1)	33	2.0 (0.2) 33	* <i>p</i> < 0.03
Typical pain intensity	2.4 (0.1)	33	2.2 (0.1) 33	† <i>p</i> < 0.02
McGill no. of words chosen	12.2 (1.0)	33	11.2 (1.1) 33	
McGill total score	29.0 (2.6)	33	24.7 (2.6) 33	* <i>p</i> < 0.01
VAS pain intensity	71.6 (3.6)	32	54.4 (5.0) 32	* <i>p</i> < 0.001
Eight word pain intensity scale	5.0 (0.3)	33	4.6 (0.3) 33	† <i>p</i> < 0.005
VAS pain relief	10.6 (2.5)	33	28.2 (5.4) 32	* <i>p</i> < 0.001
Mood	37.6 (5.0)	33	43.8 (4.9) 32	
Sleep	1.1 (0.2)	15	1.7 (0.6) 16	
Global for treatment	0.3 (0.1)	33	0.9 (0.2) 33	* <i>p</i> < 0.001

*Significant difference (Wilcoxon signed-ranks matched-pairs test) from placebo.

†Significant difference (Wilcoxon signed-ranks matched-pairs test) from baseline.

Table 3. Preference and follow-up.

No.	Preference	Significance amitriptyline vs placebo	Follow-up; months	Final dose; mg	Comment
1	none	no			
3	amitriptyline	no	8	75	continue, still doing well
4	amitriptyline	no	3	25	complete pain relief, adverse effects
5	none	yes	9	25	75 mg gave adverse effects
6	none	no	1	100	no relief, stopped
7	none	no			
8	none	no			
9	none	no	2	150	stopped, no relief and adverse effects
10	amitriptyline	yes	8	50	75 mg made no difference
12	none	yes			
15	amitriptyline	yes	12	20	? relief, drowsy in morning
16	none	no	4	100	discharge, better
17	none	no			
18	amitriptyline	yes	14	10	very good relief
19	none	no			
20	amitriptyline	yes	12	25	50 mg no more effect
21	amitriptyline	yes	11	50	100 mg gave adverse effects
24	amitriptyline	yes	3	40	stopped, no relief and adverse effects
26	amitriptyline	yes	5	150	stopped, no relief and adverse effects
27	none	no			
28	amitriptyline	no	2	100	changed to dothiepin
29	amitriptyline	no	12	100	moderate relief, foul taste
30	amitriptyline	no	10	150	good pain relief, dry mouth
31	amitriptyline	yes			
32	amitriptyline	yes	8	25	stopped due to weight gain
33	none	yes			
34	none	yes	10	75	
35	placebo	yes	11	25	good relief no adverse effects
36	amitriptyline	no	9	150	good relief
37	placebo	no	2	25	very drowsy in morning
38	amitriptyline	no	8	100	good relief, adverse effects
39	amitriptyline	no	3	45	stopped, no relief and adverse effects
40	none	no			
		Median	8	50	
		range	1-14	10-150	

Significance amitriptyline vs placebo refers to within-patient daily pain relief.

analogue) for amitriptyline compared with placebo. The global score for treatment was significantly higher for amitriptyline compared with placebo.

After 3 weeks' treatment with amitriptyline, typical pain intensity, McGill total score, visual analogue pain intensity and eight-word pain intensity scale were all significantly lower than at baseline. There were no such significant reductions after placebo treatment. Seventeen patients expressed a preference for the amitriptyline treatment, 14 had no preference, two chose placebo. Of the 14 patients whose daily pain relief scores were significantly higher with amitriptyline, nine preferred amitriptyline, one placebo and four had no preference (Table 3). The only significant order effect detected was higher mean pain relief scores on the first week of placebo treatment in those patients who had amitriptyline for their first treatment (1.1 ± 0.3 vs 0.5 ± 0.2 , $F_{4,5}$, $p = 0.05$). The post-study follow-up information obtained at routine clinic visits is given in Table 3. Twenty-three patients continued to take amitriptyline for a median of 8 months (range 1–14), with a median dose of 50 mg (range 10–150).

Adverse effects

There was no significant within-patient difference in the mood scale for the two treatments (Table 2), and no significant difference for sleep. Neither mood nor sleep were significantly changed from baseline by either amitriptyline or placebo. A significantly greater number of patients had adverse effects with amitriptyline compared with placebo (Table 4), and there was a significantly higher incidence of drowsiness and of dry mouth.

Discussion

The overall result of this study is that 25 mg of amitriptyline produced statistically significant analgesia compared with placebo. This benefit occurred within the first week (Fig. 2), as has been found in another antidepressant pain study [4], but not in all [3, 10]. The benefit was shown compared with both placebo and baseline. When daily pain relief scores for the two treatments were compared, 14 patients had significantly higher pain relief scores with amitriptyline compared with placebo.

No significant effect on mood was found with this dose of amitriptyline, either compared with baseline or with placebo. This separation of the mood and analgesic effects has been seen in chronic pain patients before [9, 10]. This study tested only one (low) dose, without preliminary kinetic or effect dose-ranging. The significant analgesic effect at this low dose, together with the supporting follow-up information showing a median dose on follow-up of 50 mg, and taken with the lack of evidence of mood change, lend credence to the idea that the analgesic and mood effects are separable, and that the dose-response curve for analgesic effect of antidepressants is shifted to the left of the curve for antidepressant effect [14]. No significant sleep effect was found at the amitriptyline dose tested. This is contrary to clinical experience, where many patients report an improvement, and to controlled studies [4]. This may be because of the low dose of amitriptyline used rather than failure of the sleep scale.

Most patients in this study were able to tolerate amitriptyline at the dose tested, but the disparity between signifi-

Table 4. Adverse effects.

	Amitriptyline (n = 33)	Placebo (n = 33)
Drowsy	12*	1
Nausea	2	4
Diarrhoea	0	1
Headache	3	6
Dry mouth	9*	2
Thirst	3	1
Tired	2	1
Foul taste	2	0
Constipation	1	1
Itch	1	1
Weight gain	1	1
Nightmares	1	0
High blood sugar	1	0
Vomit	1	0
Sweaty	0	1
Depressed	0	1
Felt ill	0	1
Dizzy	0	1
Total adverse effects reported	39	23
Total number of patients reporting adverse effects	23*	15
No. of patients reporting > 1 adverse effect	10	5
No. of patients reporting > 2 adverse effect	3	3
No. of patients with adverse effects with both treatments		8

*significant difference $p < 0.05$ Chi-square.

cant relief and preference, only nine preferences for amitriptyline among the 14 patients who had significantly greater relief whilst taking amitriptyline compared with placebo (Table 1), may reflect an adverse balance between efficacy and adverse effects, at least for the five patients who had greater relief but did not prefer amitriptyline. The follow-up data show that a number of patients tried a higher dose; some had no greater analgesia, others found increased adverse effects and reduced the dose back to a tolerable level. Whether toxicity is improved without loss of efficacy by using more selective antidepressants remains to be answered [4, 18].

The admission criterion for this study was the clinical decision to start antidepressant prescription for analgesic purposes in a heterogeneous group of chronic pain patients. This is necessarily less precise than the study of antidepressant analgesic activity in a particular pain syndrome. Of previous antidepressant studies in heterogeneous chronic pain patients, two produced significant benefit compared with placebo [11, 12], and one showed significant benefit at 2 and 4 weeks, but not at 6 weeks [13]. The suggestion has been made that the effect is small although statistically significant [12]. Judging the extent of clinical benefit from pain scores may be misleading. In chronic pain states it is not unusual for pain intensity scores to show little change from an intervention, although pain relief scores show positive benefit. This subtlety is apparent in this paper, where there was little mean percentage change in current pain intensity, but 67% greater pain relief at the end of the third week with amitriptyline compared with placebo. Although the design did not include a washout period the only order effect detected was in the first week of the second treatment, when patients

who had changed from amitriptyline to placebo had higher relief scores than those who started with placebo.

The overall results showed that the clinical decision to prescribe amitriptyline would have been justified even at this low dose in about half the cases (14 patients with significantly better relief on amitriptyline compared with placebo) (Table 3). Analgesic effect occurred quickly suggesting that clinical strategies to find the optimum dose could use 1 week as the period at which the decision to change dose could be made. For the future we should like to be more accurate in our prediction of individual response [19], and to have a clearer concept of the dose-response.

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Mivacurium chloride: a study to evaluate its use during propofol–nitrous oxide anaesthesia

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Summary

We assessed the neuromuscular and cardiovascular effects of mivacurium chloride, a neuromuscular blocking agent, in 33 patients during propofol–nitrous oxide anaesthesia. Neuromuscular function was assessed with supramaximal stimuli of the ulnar nerve, using surface electrodes at the wrist, with repeat trains of four. Mivacurium given as a bolus of 0.15 mg.kg^{-1} ($\text{ED}_{95} \times 2$) was found to be haemodynamically stable. Intubating conditions assessed at 2 and 2.5 min were either good or excellent. All patients developed a block of 100% in a mean (SD) time of 105 (34) s. There were mean (SD) intervals of 12 (2.4) min before the reappearance of the first twitch of the train of four (T1) following the bolus dose, and 15.8 (3.1) min for the T1 to reach 25% of its control value (TC). Seventeen patients received an infusion of mivacurium to maintain neuromuscular blockade (T1:TC 10–20%) with a mean (SD) infusion rate of $6.9 (2.2) \mu\text{g.kg}^{-1}.\text{min}^{-1}$. Recovery from neuromuscular blockade was assessed with spontaneous offset or augmented with edrophonium following either the initial bolus or an infusion. Following a bolus it took a mean (SD) of 26.2 (3.7) min for the fourth twitch of the train of four (T4):T1 ratio to reach 0.7. In patients receiving an infusion with spontaneous offset it took a mean (SD) time of 12.0 (2.2) min to reach the T4:T1 ratio of 0.7 from a T1:TC value of 8.8. Edrophonium significantly decreased the recovery time in both groups. It took a mean (SD) time of 5.4 (2.2) min to reach a T4:T1 of 0.7 from a T1:TC of 17.1% in the group receiving an infusion and 4.0 (0.7) min for the T4:T1 to reach 0.7 from a T1:TC value of 14.3% in the patients receiving a bolus only. We conclude that mivacurium is a suitable agent for neuromuscular relaxation and may have advantages over existing agents.

Key words

Antagonists, neuromuscular relaxants; edrophonium.

Monitoring; neuromuscular blockade.

Neuromuscular relaxants; mivacurium chloride.

Mivacurium chloride (BW B1090U) is a non-depolarising neuromuscular blocking agent which is currently being evaluated in clinical trials. It is a benzylisoquinolinium diester compound. The basic benzylisoquinolinium structure provides good potency and the two ester linkages allow for rapid hydrolysis by plasma cholinesterase [1]. These features suggest that when administered as a bolus it should have a reasonably rapid onset of action, which is of short duration, following which neuromuscular blockade may be maintained with an infusion. Several clinical studies have supported these theoretical expectations in both adults [2–10] and children [11] using various bolus doses of mivacurium and anaesthetic regimens.

Our study was designed to assess tracheal intubating conditions, onset and recovery of neuromuscular function

and cardiovascular effects after a bolus dose of mivacurium of 0.15 mg.kg^{-1} ($\text{ED}_{95} \times 2$) during anaesthesia induced and maintained with propofol supplemented by nitrous oxide. We also assessed maintenance of neuromuscular blockade using an infusion of mivacurium in some patients and the recovery of neuromuscular function either spontaneously or following the administration of edrophonium to antagonise the residual neuromuscular blockade.

Methods

With a clinical trials exemption certificate and local hospital ethics committee approval, 33 patients gave written informed consent to participate in the study.

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Table 1. Four different groups for randomisation.

Group	Time of intubation (min)	Recovery or reversal from neuromuscular block
A1	2	Edrophonium
A2	2	Spontaneous
B1	2.5	Edrophonium
B2	2.5	Spontaneous

The patients were aged between 18 and 55 years, ASA category 1 or 2, within 20% of their ideal body weight, male or female, of nonchild-bearing potential, having minor or moderate risk elective surgical procedures requiring tracheal intubation. Patients who gave a personal or family history of malignant hyperthermia, or unusual sensitivity to neuromuscular blocking agents were not studied, neither were patients with a history of chronic drug abuse, exposure to antidepressant drugs within one week, antibiotics (except erythromycin or penicillins), antihistamines, or other drugs known to affect neuromuscular function within 48 h of the study. Patients with a history of asthma (defined as having had an asthma attack within the last 3 years) were also not studied. The patients were allocated at random to one of four groups A1, A2, B1 or B2 (Table 1) according to the time of intubation and the mode of recovery from neuromuscular blockade.

The patients were unpremedicated. After intravenous access was established anaesthesia was induced with alfentanil $10 \mu\text{g.kg}^{-1}$ and a sleep dose of propofol. Anaesthesia was maintained with a propofol infusion ($6\text{--}9 \text{ mg.kg}^{-1}.\text{h}^{-1}$) supplemented by 66% nitrous oxide in oxygen.

Patients were monitored throughout with an electrocardiograph, pulse oximeter, intermittent noninvasive systemic arterial blood pressure measurements and capnography. Neuromuscular function was monitored with supramaximal stimulation of the ulnar nerve, using surface electrodes at the wrist, with repeat trains of four (TOF; 2 Hz for 2 s at 10 s intervals) using a Biometer Myotest DBS nerve stimulator. The response of the adductor pollicis muscle was measured with a force transducer and recorded using a myograph 2000, the arm being placed on an arm board. The amplitude of the first twitch of the TOF (T1) response after administration of mivacurium was expressed as a percentage of the control (baseline) response (TC). The initial dose of mivacurium given after a stable baseline recording was obtained. Monitoring of neuromuscular function was continued after the end of surgery until the T4:T1 ratio reached 0.7.

After establishing neuromuscular monitoring all patients received mivacurium 0.15 mg.kg^{-1} given over 15–30 s. At 2 min (groups A1 and A2) or 2.5 min (groups B1 and B2) following the bolus dose of mivacurium, the patient's trachea was intubated and the intubating conditions assessed (Table 2). The patient's lungs were ventilated to maintain normocapnia. Following recovery of T1:TC to 10–20% an infusion of mivacurium, initially set at $10 \mu\text{g.kg}^{-1}.\text{min}^{-1}$ was started. The infusion rate was adjusted by approximately $1 \mu\text{g.kg}^{-1}.\text{min}^{-1}$ at intervals of 3 or more min with the aim of maintaining T1:TC at 10–20%. At the end of surgery, neuromuscular function was allowed either to recover spontaneously (groups A2 and B2) or residual neuromuscular block was antagonised with edrophonium 0.5 mg.kg^{-1} given with atropine 1.2 mg

Table 2. Criteria used to assess intubating conditions.

Grade	Description
1	Excellent (easy passage of the tube without coughing).
2	Good (passage of tube with slight coughing and/or bucking).
3	Poor (passage of tube with moderate coughing and/or bucking).
4	Not possible (vocal cords tightly adducted).

(groups A1 and B1). Following return of T4:T1 to 0.7 the propofol infusion was switched off and the patient's trachea was extubated once spontaneous ventilation had been established. The patient was then transferred to the recovery room.

For each patient, following the initial dose of mivacurium, the times for maximal blockade of the neuromuscular function to occur were recorded and the times from the initial bolus of mivacurium to the beginning of recovery and to T1:TC values of 5, 10 and 25% were recorded. For assessment of recovery there were four groups depending on whether an infusion of mivacurium was needed or not and on whether recovery was spontaneous or edrophonium augmented. At the end of surgery, following the administration of edrophonium or having discontinued the infusion of mivacurium, the times for T1:TC to reach 25 and 75% and for the T4:T1 ratio to reach 0.7, were measured and the 25–75 recovery index was calculated.

To assess the haemodynamic effects, the systemic arterial blood pressure and heart rate were recorded before induction, after induction (before giving mivacurium) and then every minute for 5 min following the bolus dose of mivacurium. It was recorded every 5 min thereafter in keeping with normal clinical practice.

The results were analysed using mean, standard deviation, unpaired Student's *t*-test and Chi-squared tests where appropriate. Statistical significance was assumed at $p < 0.05$.

Results

Data has not been included on one patient whose body weight was less than 20% of his ideal. In three others' data on final recovery from neuromuscular block has been omitted as there were technical problems with monitoring neuromuscular function and the infusion of mivacurium during surgery.

The demographic data for the patients is shown in Table 3. The different groups are comparable with respect to the different intubating times and the analysis of the final recovery measures. There were 32 patients within 20% of their ideal body weight who received a bolus dose of mivacurium of 0.15 mg.kg^{-1} .

The intubating conditions at 2 min and 2.5 min are shown in Table 4. Whilst those intubated at 2.5 min had better intubating conditions compared with those intubated at 2 min this did not reach statistical significance $p < 0.1 > 0.05$ (Chi squared). The intubating conditions were always either good or excellent.

All patients developed a 100% block in a mean (SD) time of 105 (34) s (range 60–200). The initial recovery characteristics after the bolus dose are shown in Table 5.

Table 3. Demographic data of patients (mean, SD and ratio, as appropriate).

	Age (years)	Height (cm)	Weight (kg)	M:F	Induction (propofol mg.kg ⁻¹)
Total	30 (8.5)	171 (8.6)	70 (12.1)	17:15	2.5 (0.5)
Intubation 2 min	30 (7.6)	172 (9.7)	71 (8.2)	10:6	2.5 (0.5)
Intubation 2.5 min	30 (9.5)	169 (7.3)	69 (15.3)	7:9	2.4 (0.5)
Mivacurium infusion and spontaneous offset of neuromuscular blockade	36 (7.3)	168 (5.9)	68 (11.0)	3:3	2.5 (0.3)
Mivacurium infusion and edrophonium evoked recovery	31 (10.1)	172 (8.9)	71 (8.6)	7:4	2.5 (0.4)
Initial dose only plus spontaneous offset of neuromuscular blockade	26 (4.5)	173 (7.8)	76 (16.2)	4:4	2.3 (0.4)
Initial dose only plus edrophonium evoked recovery	25 (1.7)	170 (13.8)	62 (10.1)	1:3	2.6 (1.2)

The mean (SD) time to the appearance of a single twitch was 12.0 (2.4) min and for the T1:TC to reach 25% took a mean (SD) time of 15.8 (3.1) min.

Of the 19 patients who received mivacurium by infusion two were omitted from the analysis due to technical problems with the infusion. The remaining 17 received a mean (SD) dose of 6.9 (2.2) $\mu\text{g.kg}^{-1}.\text{min}^{-1}$ over a mean period of 36.2 min (range 8–144). The actual mean (SD) infusion rate at the time of administering edrophonium or on discontinuing the infusion to allow for spontaneous recovery was 5.3 (1.9) $\mu\text{g.kg}^{-1}.\text{min}^{-1}$. In the patients who received an infusion of mivacurium, the initial regimen of 10 $\mu\text{g.kg}^{-1}.\text{min}^{-1}$ returned the T1:TC ratio to a value of 0% and in some cases surgery had finished before a stable infusion regimen was established and a T1:TC value of 10–20% achieved. In addition there were some patients in whom a reduction of 1 $\mu\text{g.kg}^{-1}.\text{min}^{-1}$ in the infusion rate, with a T1:TC value near 10%, resulted in over-correction. For these reasons the T1:TC value of 10–20% aimed for in the protocol was not always achieved by the end of surgery. However, there was no statistical difference between the groups who received an infusion in the T1:TC value at the time of stopping the infusion of mivacurium or of administering the edrophonium.

The times taken for the recovery parameters recorded in the four different groups following edrophonium or the stopping of the infusion of mivacurium are shown in Table 6, together with the T1:TC% at the time the infusion was switched off or the bolus of edrophonium given. The patients who received edrophonium to augment their recovery from neuromuscular blockade made a significantly faster recovery compared with those allowed to

make a spontaneous recovery ($p < 0.001$ comparing evoked and spontaneous recovery in patients having a bolus only, and $p < 0.02$ in those receiving an infusion comparing spontaneous and evoked recovery). The 75–25 recovery index was also significantly faster in the patients who received an infusion of mivacurium followed by edrophonium compared with those recovering spontaneously ($p < 0.001$). There was no difference in the 75–25 recovery index in the patients who received a bolus dose of mivacurium only.

The heart rate and mean arterial blood pressure decreased after the induction of anaesthesia, but following the bolus injection of mivacurium there was haemodynamic stability. After laryngoscopy there was a rise in both cardiovascular measurements at 3 min (Figs 1 and 2).

In no patient was there any adverse reaction related to mivacurium and in no patient was there any clinical evidence of histamine release as evidenced by cardiovascular changes or cutaneous flushing. There were no postoperative problems.

One patient was erroneously entered into the study at less than 20% of his ideal body weight (height 178 cm, weight 45 kg). In this individual intubation was performed at 2 min (T1:TC 23%) after the bolus dose of mivacurium and was considered poor (moderate coughing and bucking). The patient developed a maximum block of T1:TC 3% which occurred 240 s after the initial bolus dose. Neuromuscular function recovered to a T1:TC value of 5% at 6.2 min and 25% at 8.8 min after the initial bolus. An infusion of mivacurium was started with which there were some technical problems; however, when the infusion

Table 4. Intubating conditions at 2 and 2.5 min after the bolus dose of mivacurium. Numbers of patients.

Grade	2 min	2.5 min
1	5	10
2	11	6
3	0	0
4	0	0

Table 5. Neuromuscular effects and recovery characteristics following the bolus dose of mivacurium.

Time for	Mean	SD	Range	n
100% block after bolus dose; s	105	34	60–200	32
Start of recovery; min	12.0	2.4	8.0–19.0	31
T1:TC = 5%; min	13.4	2.6	8.5–21.5	31
T1:TC = 10%; min	14.4	2.7	9.0–23.0	31
T1:TC = 25%; min	15.8	3.1	11.8–25	31

Table 6. Final neuromuscular recovery characteristics of the four groups (mean, SD plus range for 25–75% index).

Time (min) for recovery parameters to occur	I & S (n = 6)	I & E (n = 11)	B & S (n = 8)	B & E (n = 4)
T1:TC = 25%	4.6 (2.6)	0.4 (0.3)	17.7 (3.6)	0.4 (0.3)
T1:TC = 75%	7.3 (2.3)	1.1 (0.7)	23.0 (4.2)	3.3 (1.6)
T4:T1 = 70%	12.0 (2.2)	5.4 (2.2)	26.2 (3.7)	4.0 (0.7)
25–75% index	2.6 (0.8) (1.8–3.8)	0.8 (0.7) (0.1–2.1)	5.3 (2.2) (2.0–9.0)	2.9 (1.3) (1.5–4.4)
T1:TC at end of infusion or intravenous edrophonium	8.8 (0–20)	17.1 (5–33)		14.3 (10–25)
Infusion rate; $\mu\text{g.kg}^{-1}.\text{min}^{-1}$	6.4 (1.6)	4.7 (1.9)		

I, mivacurium infusion; B, mivacurium bolus; S, spontaneous offset; E, edrophonium evoked recovery.

was switched off the T1:TC value was 55% and it took 9.1 min for the T4:T1 ratio to reach 0.7 with spontaneous offset of neuromuscular blockade.

Discussion

The aim of this study was to assess the neuromuscular and cardiovascular effects of mivacurium following a bolus dose of 0.15 mg.kg^{-1} ($\text{ED}_{95} \times 2$). This dose was chosen since higher doses of mivacurium (0.2 and 0.25 mg.kg^{-1}) have been associated with untoward cardiovascular effects, in particular hypotension caused by the vasodilation following histamine release [12]. The bolus was given over 15–30 s as this has been shown to reduce the cardiovascular effects associated with any histamine release [5]. In our study there was good haemodynamic stability following the bolus of mivacurium. There were rises in heart rate and mean arterial blood pressure 3 min after the administration of the bolus of mivacurium due to the stimulus of laryngoscopy and tracheal intubation.

Inhalational anaesthetic agents have been shown to potentiate the action of neuromuscular blocking drugs including mivacurium [3, 5, 6, 9] compared with anaesthetic regimens based on thiopentone and opioids. In our study, patients received a different anaesthetic regimen, using propofol for both induction and maintenance of anaesthesia, which is not known to have an effect on the neuromuscular junction.

There was a tendency for intubating conditions at 2.5 min to be better than at 2 min although this did not reach statistical significance. There were four patients who had not reached a 100% block at the time of intubation, of

which three were grade two and one grade one. The finding that coughing and bucking can occur despite apparent 100% block, as measured at the ulnar nerve, demonstrates that different muscle groups appear to have different affinities for neuromuscular blocking drugs, with a more profound effect appearing on the muscles of the hand than on the muscles of the larynx and the diaphragm.

The initial recovery profile found in our study is similar to that found by other workers with a bolus of 0.15 mg.kg^{-1} , despite differing anaesthetic regimens [3, 5–8, 10] so that 14 min after the bolus, neuromuscular function had recovered to a value of T1:TC of 10%; this compared with a value of 40 min following a bolus of atracurium of 0.5 mg.kg^{-1} [7].

The decision to start an infusion of mivacurium or not was at the discretion of the responsible anaesthetist and was based upon the expected duration of surgery, for instance a number of patients having simple extraction of wisdom teeth did not require prolonged neuromuscular paralysis. This is why there are different numbers in the four groups for the final recovery characteristics. The initial infusion rate of $10 \mu\text{g.kg}^{-1}.\text{min}^{-1}$ was too great in all patients and needed adjusting downwards. The mean given was $6.8 \mu\text{g.kg}^{-1}.\text{min}^{-1}$ with a mean rate at the time of giving edrophonium and of switching off the infusion of $5.3 \mu\text{g.kg}^{-1}.\text{min}^{-1}$. This is similar to values found by other workers [4, 5, 10]. In several patients a stable infusion regimen, with the 10–20% T1:TC ratio aimed for, was not achieved. In some cases neuromuscular function had not recovered from the initial high infusion rate and in others variability in the response with time led to a greater T1:TC ratio than desired (10–20%). However, we were trying to

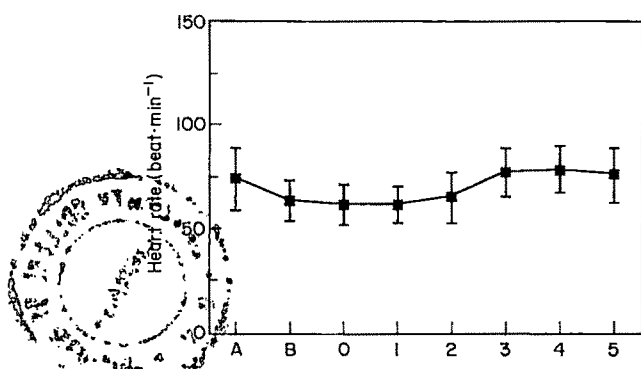


Fig. 1. Changes in heart rate at the times shown (A, baseline before induction of anaesthesia; B, after induction of anaesthesia; O, time mivacurium given, 1–5 min after giving mivacurium).

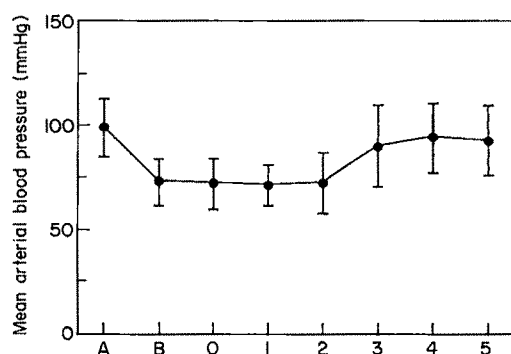


Fig. 2. Changes in mean arterial blood pressure at the times shown in the legend to Fig. 1.

simulate normal clinical practice for the use of mivacurium and did not follow an extremely rigid protocol. We would now suggest a lower starting infusion rate $5\text{--}6\text{ }\mu\text{g.kg}^{-1}.\text{min}^{-1}$ especially in the clinical setting where monitoring of neuromuscular function may be less sophisticated. Other workers have suggested that there is a wide variation in requirements for infusion regimens with mivacurium [10]. As some patients in our study did not reach a stable level we feel that it is not possible to draw any conclusions about this from our results although it does seem likely that this will be the case. This emphasises the need for neuromuscular monitoring in all patients who receive neuromuscular blocking drugs [13].

The group in our study who were allowed to make a spontaneous recovery from a bolus had recovered to a T4:T1 ratio of 70% after 26.2 min compared with 4 min from a mean T1:TC of 14.3% in the group receiving edrophonium. This suggests that edrophonium antagonism of the neuromuscular block only speeds recovery by approximately 7 min (14.4 min to recovery from bolus of T1:TC of 10% plus 4 min after edrophonium administration compared with 26 min after spontaneous offset). This rapid recovery with mivacurium has led Savarese and colleagues to question the need for the administration of any anticholinesterase with mivacurium since it does not clinically speed recovery to a significant extent [14]. The amount of time saved following an infusion of mivacurium comparing spontaneous offset with evoked recovery using edrophonium was also of the order of 7 min (12.0 and 5.4 min respectively). In contrast, recovery from atracurium and vecuronium, compared with mivacurium after an infusion to maintain a block of 90–99%, took twice as long to recover to a T1:TC of 25% and the 25–75 recovery index was significantly longer with both atracurium and vecuronium (infusion off — 25%: atracurium 12.5 min; vecuronium 12.7 min; mivacurium 5.7 min and 25–75 recovery index: atracurium 10.9 min; vecuronium 13.8 min; mivacurium 6.4 min) [8].

In the low weight patient who was erroneously admitted to the study the mivacurium did not produce as profound a block as in the other patients; this could have been predicted as his lean muscle mass is likely to be a proportionately greater amount of his total body mass than in patients who are within 20% of their ideal body weight. Similarly, obese patients are likely to need less than the recommended dose as their lean muscle mass is likely to be proportionately lower than the total body weight.

In conclusion, we found that at a bolus dose of 0.15 mg.kg^{-1} mivacurium produced good intubating conditions at 2 or 2.5 min with no untoward cardiovascular effects. In all patients there was an initial fast recovery, following which neuromuscular blockade could be readily maintained with an infusion. The faster recovery, which is consistently observed with mivacurium when administered either as a bolus or via infusion both with spontaneous offset and augmented recovery, would make it a suitable agent for producing neuromuscular blockade for short and long surgical procedures requiring intubation. The faster

offset of mivacurium, compared with other competitive neuromuscular blocking drugs, may make it a useful agent in day case surgery.

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An analogue approach to audit in anaesthesia

P. V. SCOTT, R. M. HADEN, C. A. PINNOCK, H. S. BRIDGE AND A. J. LAWRANCE

Summary

The predicted outcome of anaesthesia and surgery was compared with the immediate outcome in 508 patients by means of two 100 mm linear analogue scales. The results were used to obtain a statistically based rule by which the anaesthetist may consistently select three groups of patients for audit: group 1, patients in whom immediate outcome of anaesthesia and surgery is worse than predicted; group 2, patients whose outcome is better than predicted; and group 3, the remaining patients. The rule, which is simply adapted to departmental audit, does not necessarily need a computer but is suited to the computer as it is numerically based.

Key words

Anaesthesia; audit.

Measurement; linear analogue scale.

Which patients should the anaesthetist select for clinical audit [1]? Deaths are self-selecting and there is no dispute about the end point, but mortality surveys, especially when buttressed by informed autopsy reports [2], have the power to change clinical practice [3–8]. With regard to morbidity there is often dispute and the question is how to resolve it [9–13].

One answer to that question, and the aim of this study, may be to obtain a rule by which an individual anaesthetist can, with confidence and consistency, select three groups of patients for audit: group 1, patients in whom the immediate outcome from anaesthesia and surgery is worse than predicted; group 2, patients whose outcome is better than predicted; and group 3, the remaining patients. Groups 1 and 2 represent extremes of outcome.

The study was based on the completion, in 508 patients, of two 100 mm linear analogue scales (LAS). One scale signified the predicted outcome of anaesthesia and surgery, viewed as a composite entity, while the other signified the perceived outcome.

Method

A pilot study in 156 patients was conducted to gain experience in the use of the linear analogue scale [14] and in the handling of data. Thereafter, during three consecutive months, 508 unselected patients admitted to a small district general hospital and who underwent surgery were studied

prospectively. They were visited on the ward or in the accident and emergency department by one of five anaesthetist assessors and were later anaesthetised by that assessor. Patients who received extradural analgesia during labour were not studied.

Assessment before operation (prediction)

Patient assessment was made in the customary manner. A global view was taken of the risks of anaesthesia and surgery on the basis of clinical intuition and experience, the medical history, physical examination, the results of investigations and the demands of pre- and postoperative care. The anaesthetist discussed the patient with the accompanying nurse, with surgical staff and sometimes with relatives. Suitable anaesthetic techniques were reviewed. The patient's morale, the surgeon's skill, the ASA and the Mallampati classifications [15, 16], the site and nature of the operation, the timing of surgery, and the Confidential Enquiry into Peri-operative Deaths (CEPOD) classification of degree of urgency [4] were all deemed relevant.

The prediction assessment, after summation into a global score, was recorded on a 100 mm linear analogue scale (termed 'x'). The predictive LAS represented 'best possible predicted outcome of anaesthesia and surgery' at one end ($x = 0$ mm) and 'worst possible predicted outcome of anaesthesia and surgery' at the other ($x = 100$ mm).

Each unmarked horizontal 100 mm line was drawn

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across the middle of a headed sheet of A4 paper. The heading sought demographic data.

Assessment after operation (immediate outcome)

Before discharge from the recovery ward the patient was assessed in the anaesthetist's customary manner and a second 100 mm LAS marked (termed 'y'). The outcome LAS represented 'best possible perceived outcome of anaesthesia and surgery' at one end ($y = 0$ mm) and 'worst possible perceived outcome of anaesthesia and surgery' at the other ($y = 100$ mm). Outcome was judged, without reference to the predictive LAS, on events which occurred during induction of general, regional, or local anaesthesia, or their combination; by events which occurred during maintenance of anaesthesia; by any observed surgical problems; and by the quality of recovery from anaesthesia.

Statistical analysis

Descriptive rather than inferential methods were used [17–19]. The paired values of x and y for each patient were measured to the nearest mm by one investigator and displayed by scatter plots as coordinates x , y . The spread and symmetry of the corresponding derived quantities ($x + y$) were displayed as 'box and whisker' plots giving the median, quartiles, and range [18] (Fig. 1). High outlying values of ($x + y$), 'the outliers', were sought; by convention, these are to be found 1.5 interquartiles beyond the upper quartile of the data (see Appendix). Mathematical tools to generate p values were not used since the one aim was to identify the outliers.

Results

Demographic data are given in Table 1 and the interquartile range of ($x + y$) in Table 2. Differences in the numbers

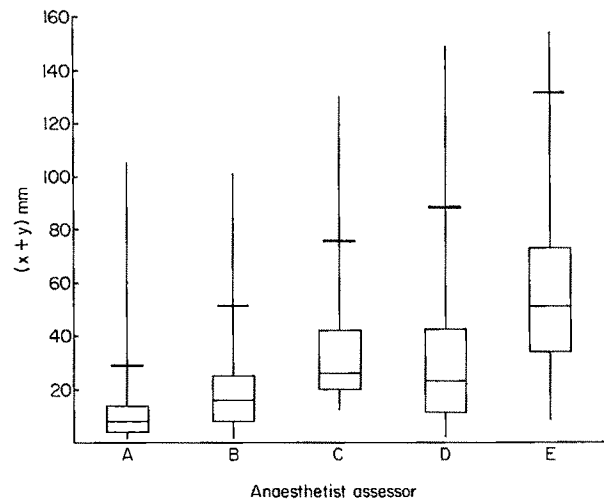


Fig. 1. 'Box and whisker' plots [18] of ($x + y$) for the five anaesthetist assessors. The large rectangles or 'boxes' (of length H , the interquartile range, containing the middle 50% of the data) are divided by the median into smaller rectangles, whose extremes are the upper (25%) and lower (25%) quartiles. The ends of the vertical lines ('whiskers') are the limits of the range. The bars are 1.5 times the interquartile range beyond the upper quartile. Values beyond the bars are the outliers, or patients suited to audit. See Appendix.

of patients studied by each anaesthetist are partly explained by case mix. Where two or more anaesthetists were present during operation only one (the original assessor) was entitled to mark the linear analogue scale.

The coordinates x , y for the five anaesthetist assessors are shown in Figure 2 (a–e). The 45° dashed diagonal represents the line of identity: $x = y$ (prediction = outcome). Neither x and y nor the derived quantity ($x + y$) were symmetrically distributed. High outlying values of

Table 1. Demographic data.

Assessor (grade)	Years in specialty	Patients studied		
		n	M/F%	Age; years: range (mean)
A (Cons)	12.0	87	42/58	< 1–91 (37)
B (Cons)	24.0	123	31/69	< 1–90 (44)
C (Cons)	13.0	108	45/55	2–87 (57)
D (Reg)	5.0	98	43/57	1–88 (41)
E (SHO)	0.3	92	31/69	5–84 (45)
[Department]		[2160]	[35/65]	[< 1–102 (46)]

Cons, consultant anaesthetist, Reg, registrar, SHO, senior house officer. [Department] = work load of Department of Anaesthesia during the study.

Table 2. Interquartile values of ($x + y$) to nearest mm, where x = predicted and y = perceived outcome of anaesthesia and surgery.

Assessor (n)	Median (range)	Interquartile range (H)		1.5H + UQ (Z)
		LQ–UQ	H	
A (87)	8 (2–105)	4–14	10	29
B (123)	16 (2–101)	8–25	17	51
C (108)	26 (12–130)	20–42	22	75
D (98)	23 (2–148)	11–42	31	88
E (92)	50 (8–153)	33–72	39	131

UQ, upper quartile; LQ, lower quartile; high outlying values of ($x + y$) > Z; see Discussion and Appendix.

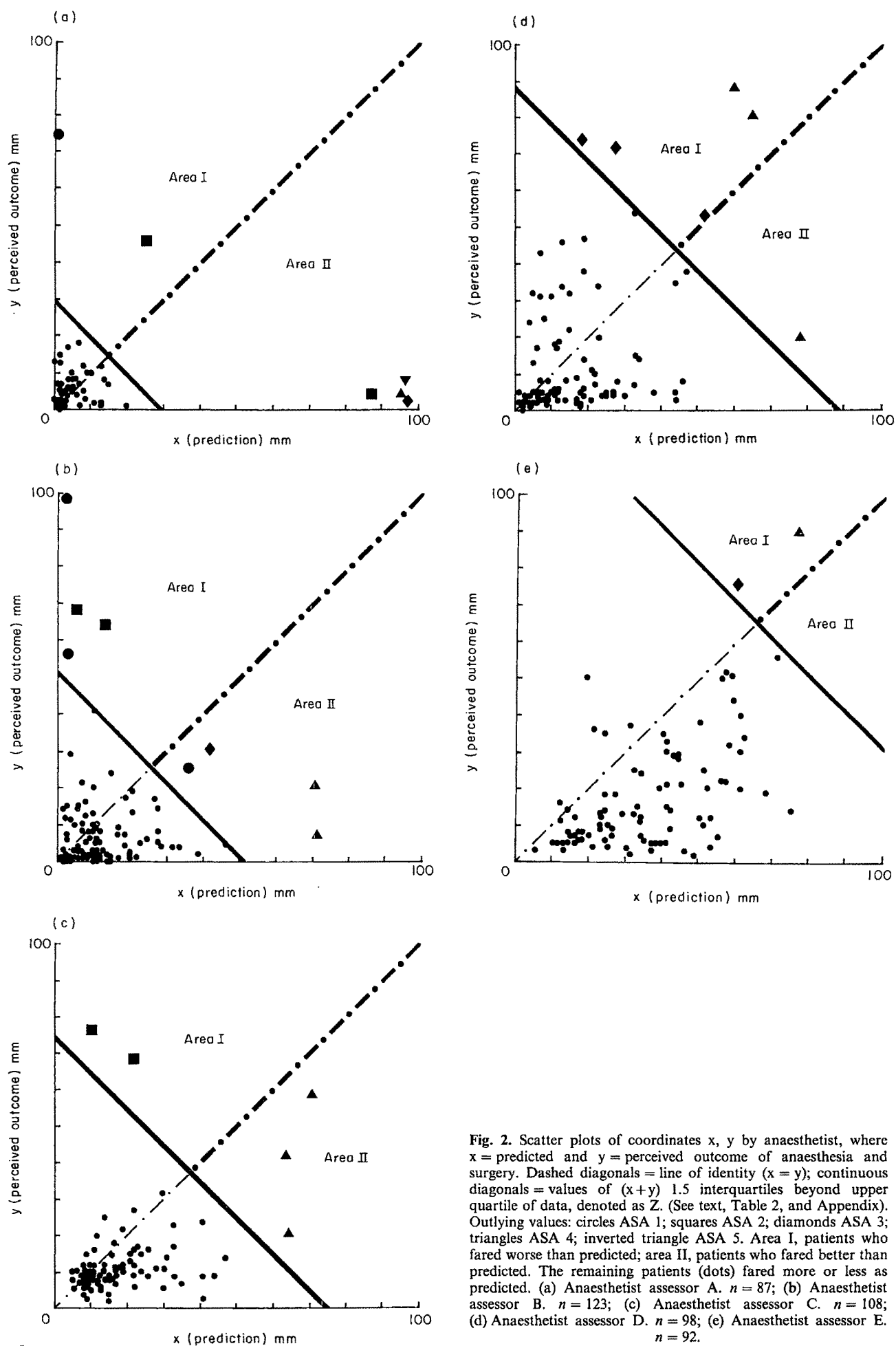


Fig. 2. Scatter plots of coordinates x, y by anaesthetist, where x = predicted and y = perceived outcome of anaesthesia and surgery. Dashed diagonals = line of identity ($x = y$); continuous diagonals = values of $(x + y)$ 1.5 interquartiles beyond upper quartile of data, denoted as Z . (See text, Table 2, and Appendix). Outlying values: circles ASA 1; squares ASA 2; diamonds ASA 3; triangles ASA 4; inverted triangle ASA 5. Area I, patients who fared worse than predicted; area II, patients who fared better than predicted. The remaining patients (dots) fared more or less as predicted. (a) Anaesthetist assessor A. $n = 87$; (b) Anaesthetist assessor B. $n = 123$; (c) Anaesthetist assessor C. $n = 108$; (d) Anaesthetist assessor D. $n = 98$; (e) Anaesthetist assessor E. $n = 92$.

($x + y$) (values beyond 1.5 interquartiles of the upper quartile of the data) lie above the bars in Figure 1 and to the right side of the continuous diagonals in Figure 2. The data were divided into three areas by the line of identity and the 1.5 interquartile (Fig. 2).

Group 1 patients (2–4% of the total) are represented by the coordinates x, y in area I (Fig. 2) and group 2 (3–5% of the total) by those in area II. Group 3 are the remainder (90–95% of the total). Groups 1–2 are the outliers. Review of each anaesthetic record by an independent observer showed that the outliers included all patients worthy of audit.

Discussion

We describe a system for obtaining a statistically based rule which identifies groups of patients suitable for individual audit in anaesthesia and which can be adapted for departmental audit. The rule is based on completion, for each patient, of two 100 mm linear analogue scales (x and y), where x = predicted outcome of anaesthesia and surgery and y = perceived outcome.

The rule: FORAY (4A — Analogue Approach to Audit in Anaesthesia)

The most suitable patients for audit are represented by the high outlying values of the derived quantity ($x + y$). Those values lie 1.5 interquartiles beyond the upper quartile of ($x + y$), denoted as Z (Table 2). Thus,

$$Z = 1.5H + UQ$$

where H = interquartile and UQ = upper quartile. (See Appendix.)

In group 1 patients ($x + y$) > Z and y > x ; the immediate outcome of anaesthesia and surgery is worse than predicted. In group 2 ($x + y$) > Z and x > y ; outcome is better than predicted. In the remaining patients (group 3, the non-outliers) ($x + y$) < Z ; outcome is more or less as predicted (Fig. 2).

Notation Each patient was identified as follows:

$$\text{FORAY} = x, y; (x + y): Z.$$

For example: in Figure 2(b) the predicted outcome x for the outlier at the top left hand corner was 2 mm, the perceived outcome y was 99 mm; ($x + y$) = 101 mm, Z = 51 mm. Hence,

$$\text{FORAY} = 2, 99; 101: 51.$$

Differences in the patterns of results between assessors may reflect case mix, experience, competence, optimism, pessimism, insight and many other factors. Too much emphasis on the differences would be insensitive [20] although the implications both for audit and research are clear. There was consistency 'within-assessor', but not 'between-assessors', and a tendency to overestimate risk. *Obiter dictum*, as a cursory remark not central to the main issue, we have formed the opinion that the differences between assessors A, B, and C were probably philosophical. Assessor A was prepared to score zero ($x = 0$) for predicted outcome and zero ($y = 0$) for perceived outcome; assessor B would score zero only for y ; and assessor C would score zero neither for x nor y . Assessor D was highly

self-critical. Results from assessor E, obtained during the first 3 months of training when accompanied by an experienced anaesthetist for 81% of cases, were reassuring; the risks of anaesthesia were nearly always overestimated.

Case histories

Case 1 A 32-year-old ASA 1 female (laparoscopic sterilisation) with a predicted outcome of 2 mm scored 99 mm for perceived outcome (FORAY: 2,99; 101: 51). External cardiac compression was required for 50 s before cardiac output could be restored after asystole during forceful manipulation of the cervix.

Case 2 An orthopnoeic respiratory cripple ASA 4 over 80 years of age (total proctocolectomy) had been reserved a bed in the intensive therapy unit for elective postoperative ventilation of the lungs. He had no need of the bed and went home 7 days after operation (72, 7; 79: 51).

Case 3 A 30-year-old manual worker, ASA 1, received conventional doses of propofol (180 mg) and alfentanil (0.5 mg). The surgeon manipulated the lumbar spine, stretched the sciatic nerves, and rapidly injected into the sacral canal 40 ml of a solution containing 20 ml bupivacaine 0.5% and methylprednisolone 80 mg in saline. Recovery from general anaesthesia, normally less than 5 min in comparable cases, was 35 min despite the use of naloxone to antagonise alfentanil (1, 75; 76: 29).

Case 4 Four days after a full-thickness anterior myocardial infarction an 89-year-old ASA 5 female with interstitial pulmonary oedema survived open reduction and internal fixation of a fractured femoral shaft (97, 8; 105: 29).

At audit: case 1 implied that patients for operative pelvic laparoscopy may benefit from prophylactic vagolysis and changed departmental advice to its trainees. Case 2, delayed 7 days by the assessor for intensive chest physiotherapy, bronchodilators, and antibiotics, and in whom general anaesthesia was combined with extradural anaesthesia, stressed the role of the anaesthetist in pre- and postoperative care. The anaesthetic and the plan for postoperative pain relief had been most carefully constructed. The patient and his wife were 'fighters'. Delayed recovery in case 3 was hard to explain but on the balance of probabilities may have been caused by a 'transient but very marked increase in intracranial pressure' [21] from too rapid injection of fluid into the extradural space [22, 23]. That practice was modified to a slower rate of injection. Case 4, which five senior anaesthetists had rejected for surgery (they had thought it to be an unjustifiable risk), showed the value of individual nerve blocks without general anaesthesia in critically ill patients and led to a useful discussion about ethics.

Comment

Case selection for audit has hitherto been arbitrary. The system we describe has the merit of consistency. Although the linear analogue is subjective so also is the ASA classification or rating of physical status [24]. ASA rating of the same patient by different anaesthetists is not consistent [25], lacks scientific precision [25] and is neither an estimate of operative risk [24, 26] nor a good predictor for mortality [4]. This study suggests that it may not be a good predictor for morbidity (Fig. 2 and case histories).

For over 70 years the linear analogue scale and its precursors have been used for the assessment of subjective phenomena [14, 19]. We would have preferred to quantify the risks and outcome of anaesthesia in a more precise way. APACHE II [27, 28], for example, has a more acceptable scientific basis but does not address anaesthetic risk; its complexity is daunting. Nor does APACHE II pretend to measure clinical acumen and a patient's morale; these are subjective phenomena not easily quantified. The 'index of risk in noncardiac procedures' is no more satisfactory [29]. There is a real problem here because elements both of the subjective and the objective are integral to any assessment of risk and outcome in medicine. Despite their subjectivity the linear analogue scales described in this study were helpful in defining both morbidity and its reverse: that is, patients who fare better than predicted. Those patients are not reviewed at audit as a routine; perhaps they should be.

A computer was not used but FORAY is suited to the computer because it is numerically based. It might be useful if FORAY were married to a computerised anaesthetic record, or to an operating theatre computer record (Taylor PA, personal communication, 1992). The applications would be limited only by the imagination of the clinician. For example, it should prove possible to follow the progress of a trainee (Fig. 1 E, Fig. 2 (e)). A computer-based study might help to identify objective criteria by which to assess prognosis, although there would remain the problem of weighting the criteria against clinical acumen and a patient's morale. FORAY might be extended to include delayed (24–48 h) and remote (> 48 h) outcome [10, 11].

There is a need for a simple, consistent and universal system, such as we describe, to select patients for clinical audit. Only then will it be possible to put the central question: 'Our results ... confirm that some factor in anaesthetic management can make a difference to outcome ... It is important to continue asking 'What?' [12].

Appendix

Derivation of FORAY

1. Take the completed linear analogue scales and measure and tabulate the paired coordinates x , y for each patient.
2. For each coordinate x , y for each anaesthetist calculate the value of $(x+y)$ and rank the values in order of magnitude.
3. Find the median value of $(x+y)$ for each anaesthetist: that is, the middle value when the observations are ranked (Fig. 3).
4. Derive the interquartile range (H , Fig. 3 (b and c)). The upper (UQ) and lower (LQ) quartiles separate off the upper and lower 25% of the values: thus the interquartile range measures the middle 50% of the data [18].
5. Take the upper quartile value (UQ) and add to it 1.5 times the value of the interquartile range (H). Let that value of $(x+y) = Z$ (Fig. 3 (b)).

Then:

$$Z = 1.5H + UQ$$

where values of $(x+y) > Z$ are the outliers suited to audit.

6. Either (i): display the results as in Fig. 3 (c) (see Fig. 1), where the bar = Z ;

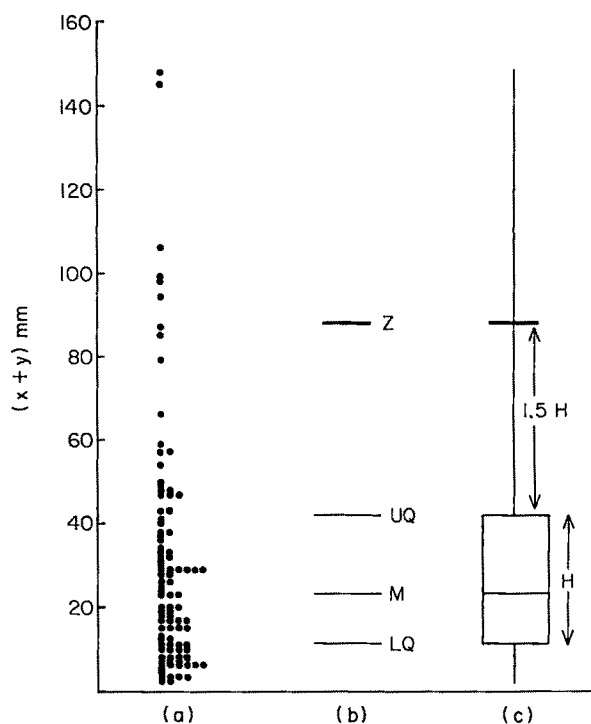


Fig. 3. Anaesthetist assessor D. (a) distribution of $(x+y)$; (b) M = median; LQ = lower quartile; UQ = upper quartile; Z = 1.5 times interquartile beyond upper quartile. (c) Box and whisker plot where H = interquartile. (After Brown RA, Swanson Beck J [18].)

or (ii): display the results as in Fig. 2, where the continuous diagonals = Z . That is,

$$\begin{aligned} \text{if } x+y &= Z \\ \text{when } x=0, Z &= y; \text{ and} \\ \text{when } y=0, Z &= x. \end{aligned}$$

7. Notation FORAY = x , y ; $(x+y)$: Z .

Note A statistics computer package such as MINITAB, STATGRAPHICS, or UNISTAT will complete steps 2–6 (i). (Suppliers: MINITAB — Clecom Ltd, The Research Park, Vincent Drive, Edgbaston, Birmingham B15 2GQ; STATGRAPHICS — Mercia Software Ltd., Aston Science Park, Love Lane, Birmingham B7 4BJ; UNISTAT — Unistat Ltd, Dept. CW, PO Box 383, Highgate, London N6 5UP.)

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Phenytoin reduces suxamethonium-induced myalgia

V. HATTA, A. SAXENA AND H. L. KAUL

Summary

A prospective, randomised trial was undertaken in 60 healthy adults to determine the efficacy of intravenously administered phenytoin in doses of 5 mg.kg⁻¹ for the prevention of suxamethonium-induced fasciculations, a rise in serum K⁺ and myalgia. This was compared with tubocurarine pretreatment and no pretreatment (control group). Phenytoin pretreatment significantly reduced myalgia from 45% (nine patients) in the control group to 10% (two patients) ($p < 0.05$). It also decreased the duration and mean intensity of fasciculations. Incidentally, phenytoin was also found to decrease significantly mean serum Na⁺ levels ($p < 0.001$) both at 5 and 20 min after administration. Tubocurarine pretreatment (3 mg) resulted in a significant decrease in fasciculations, but myalgia, which occurred in five patients, remained the same. No significant correlation was found between muscle fasciculations, postoperative myalgia and K⁺ changes, but patients with myalgia had a significant decrease in mean serum Na⁺ levels at 5 and 20 min after suxamethonium ($p < 0.01$).

Key words

Neuromuscular relaxant; suxamethonium, d-tubocurarine.

Pain, postoperative; myalgia.

Anticonvulsant; phenytoin.

Muscle, skeletal; fasciculations.

New methods are sought to prevent the undesirable effects of administration of suxamethonium, and they reflect both the importance of suxamethonium in clinical practice and the limitations of our power to prevent unwanted consequences. Many attempts have been made to develop pretreatment regimens, the most widely accepted being the use of subparalytic doses of non-depolarising drugs. However, precurarisation is found to attenuate the effect of suxamethonium, delays its onset of action and shortens the duration of block [1–5].

Recently, patients receiving chronic phenytoin therapy have been shown to be resistant to neuromuscular block induced by vecuronium [6, 7], pancuronium [8] and metacurine [9], whereas acutely administered phenytoin has been shown to prolong the duration of vecuronium-induced neuromuscular block [10].

In an experimental study [1] on cat soleus muscle, intravenous phenytoin in doses of 30 mg.kg⁻¹ has been shown to depress suxamethonium-induced fasciculations, while enhancing its blocking potency. Phenytoin increases the initial rate of block without altering its duration. Therefore, this prospective, randomised study was planned to examine the efficacy of an acutely administered, anticonvulsant dose of phenytoin to modify suxamethonium-

induced fasciculations, myalgia and rise in serum K⁺ in human subjects.

Materials and methods

Ethics committee approval was obtained for this prospective, randomised study carried out on 60 adult patients belonging to ASA class 1 and scheduled for minor surgical procedures. Patients with neurological disease or those already taking phenytoin were not studied. Informed consent was obtained. All patients were premedicated with pethidine 1 mg.kg⁻¹, promethazine 0.5 mg.kg⁻¹ and atropine 0.5 mg, 60 min before surgery. Patients were allocated at random to three groups of 20 each. Group 1 received phenytoin 5 mg.kg⁻¹ up to a maximum of 250 mg intravenously, at the rate of 50 mg.min⁻¹ 5 min before suxamethonium; group 2 received intravenous tubocurarine 3 mg 3 min before suxamethonium; group 3 was the control group and received no pretreatment. Anaesthesia was induced with thiopentone 4–5 mg.kg⁻¹ followed immediately by suxamethonium 1.5 mg.kg⁻¹. The patient's trachea was intubated after 1 min. Anaesthesia was maintained with nitrous oxide and oxygen (67:33) and halothane 1–2% with spontaneous respiration. Pancuronium or tubocur-

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arine were used to paralyse if IPPV was required. Analgesia was supplemented with incremental doses of intravenous pethidine.

Heart rate and blood pressure were recorded before and after pretreatment and every 5 min for 20 min for the purpose of the study. The ECG was monitored throughout surgery.

Following administration of suxamethonium, the presence, degree and duration of fasciculations was assessed visually on a four-point scale: 0, no visible fasciculations; 1, (mild) very fine finger tip or facial muscle movements; 2, (moderate), minimal fasciculations on the trunk and extremities; 3, (severe) vigorous fasciculations on the trunk and extremities.

Blood samples for estimation of serum K^+ and Na^+ were drawn before induction and 5 and 20 min after suxamethonium and for phenytoin levels at 5 min after its administration.

Postoperatively myalgia was assessed by enquiring about the presence, site and severity of pain immediately after transfer to the recovery room and at 24, 48 and 72 h. Assessment of pain at the surgical site was not included. The muscle pains were assessed on a four-point scale and graded as follows: 0, (none) no muscle pain; 1, (mild) generalised pain or localised pain which was tolerable; 2, (moderate) pain at numerous sites or very severe at a single site; 3, (severe) incapacitating pain.

The results are expressed as mean (SD). Statistical analysis of the data was performed by analysis of variance and Chi-squared test. The values were considered to be statistically significant at $p < 0.05$.

Results

Patient data are summarised in Table 1. The three groups were comparable in sex, age and weight distribution. In groups 1 and 3, no significant changes were observed in mean heart rate or blood pressure. In group 2, tubocurarine pretreatment caused a significant increase in mean heart rate at 5 and 10 min ($p < 0.01$ and $p < 0.05$) and a decrease in mean systolic blood pressure at 15 min ($p < 0.05$).

Incidence of myalgia in group 1 patients was 10%, as compared to 45% in group 3 ($p < 0.05$) and 25% in group 2 (Table 2). The time course of myalgia was different in the three groups (Table 3). The patients in group 1 complained of myalgia after 24 h and this was mild in intensity, not requiring an analgesic. However, in groups 2 and 3, myalgia occurred within the first 24 h after operation in the majority of the patients, with intensity ranging from mild to severe. Analgesics were required in 87% patients in both groups.

Table 1. Basic patient data from the three groups.

	1	2	3
Age; years, mean (SD)	26.2 (8.8)	27.8 (10.2)	30.5 (11.3)
Weight; kg, mean (SD)	53.6 (8.3)	55.1 (6.0)	56.5 (10.2)
Males	14	16	11
Females	6	4	9
No. of patients	20	20	20

Table 2. Distribution of patients with postoperative myalgia.

Group	Incidence of pain	Severity of pain		
		Mild	Moderate	Severe
1 ($n = 20$)	2 (10%)*	1 (5%)	1 (5%)	—
2 ($n = 20$)	5 (25%)	3 (15%)	2 (10%)	—
3 ($n = 20$)	9 (45%)	7 (35%)	1 (5%)	1 (5%)

* $p < 0.05$ (as compared to group 3).

In both groups 1 and 3, 95% patients had fasciculations, but the mean intensity and duration of fasciculations was less in group 1 as compared to group 3, although it was statistically not significant. Group 2 patients had the lowest incidence of fasciculation (25%) with the least mean intensity and duration (Table 4).

Mean serum phenytoin levels 5 min after administering the bolus dose were found to be $14.46 \mu\text{g.ml}^{-1}$. No significant change in mean serum K^+ level was observed in either of the three groups (Fig. 1). No definite correlation was found between the incidence and severity of myalgia and the occurrence of fasciculations and serum K^+ changes.

Patients in group 1 were found to have a significantly lower mean serum Na^+ level both at 5 and 20 min after suxamethonium ($p < 0.001$), whereas no significant changes were observed in the other two groups (Fig. 2). Patients with myalgia in all three groups (16 patients) were found to have significant decrease in mean serum Na^+ level at 5 min as well as at 20 min after suxamethonium administration ($p < 0.01$). One patient who had a marked increase in serum Na^+ at 20 mins (17 meq.l^{-1}) was affecting the mean significantly and was therefore excluded from the calculations of mean serum Na^+ . No patient having severe fasciculations reported severe muscle pain, while others with no fasciculations had a moderate degree of myalgia (Fig. 3). Patients pretreated with phenytoin did not appear to have a delayed recovery as compared to groups 2 and 3, although the recovery scores were not recorded for the purpose of this study.

Discussion

The syndrome of muscle aches and pains following suxamethonium administration is well recognised [11, 12]. Churchill-Davidson [13] reported an incidence of 64% and 14% in outpatients and inpatients, respectively. Various estimates since then have ranged between 0.2% to 89% [13, 14]. This range is undoubtedly a reflection of various factors which may influence the occurrence and severity of muscle pain.

The mechanism by which suxamethonium produces postoperative muscle pain is not yet known, although the drug has been in routine clinical use for nearly 40 years.

Table 3. Incidence of pain at 24, 48 and 72 h.

Group	Number of patients complaining of myalgia at		
	24 h	48 h	72 h
1 ($n = 20$)	0	2 (10%)	2 (10%)
2 ($n = 20$)	4 (20%)	3 (15%)	2 (10%)
3 ($n = 20$)	7 (35%)	5 (25%)	4 (20%)
Total	11 (18.3%)	10 (16.7%)	8 (13.3%)

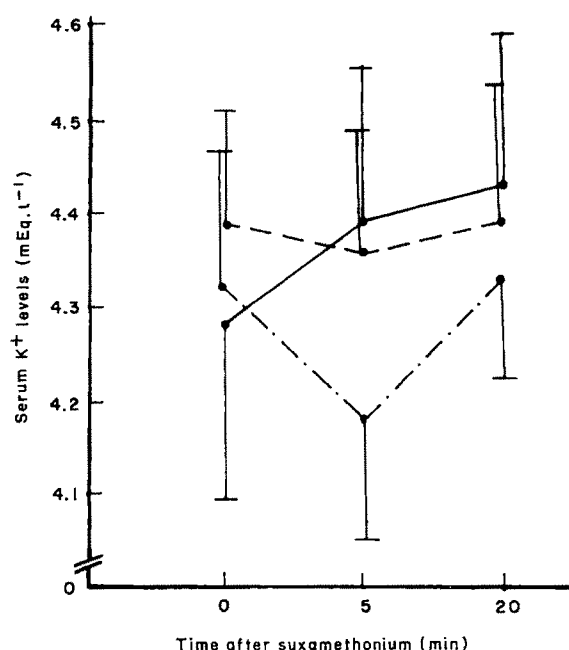


Fig. 1. Changes in serum potassium following suxamethonium injection. —●—, group 1; ····●···, group 2; —●—, group 3.

However, various theoretically possible explanations have been given [14–17].

Influx of Ca^{2+} into the muscle cells is known to enhance the speed and strength of the fasciculations and the contraction of the intrafusal muscle fibres, which increases the likelihood of spindle damage and subsequent muscle pain [17]. Phenytoin, by causing a decrease in influx of Ca^{2+} ions during depolarization, either independently or as a consequence of reduced intracellular concentration of sodium ions, may prevent muscle damage [18–21]. This can explain the significant decrease in incidence of muscle pain (10%) in our patients pretreated with phenytoin.

However, we found no reduction in the incidence of fasciculations, contrary to the result of a previous study [1]. Collier [22] has proposed that the incidence of myalgia after suxamethonium is not related to visible fasciculation but to the rate of firing of motor unit potentials recorded electromyographically. Phenytoin is known to suppress the repetitive firing of neurones, nerve terminals and muscle cells, and decrease post-tetanic potentiation evoked in cat spinal cord and sympathetic ganglia [23, 24]. It also stabilises the motor nerve terminal membrane by a nonreceptor action [18, 19]. These effects are seen at anticonvulsant levels, which can be achieved with a dose of 5 mg.kg^{-1} . In normal human volunteers, this dose produces mean plasma levels of $10 \mu\text{g.ml}^{-1}$ [25, 26]. In our study, the mean phenytoin level at 5 min was on the higher side, although within

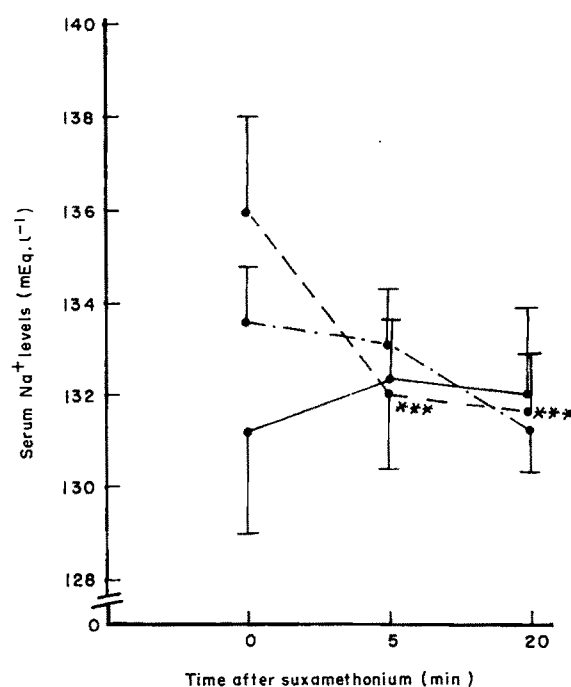


Fig. 2. Changes in serum sodium following suxamethonium injection. —●—, group 1; ····●···, group 2; —●—, group 3; *** $p < 0.01$.

the safe range, and was sufficient to decrease myalgia. It remains to be seen if phenytoin will be effective for myalgia suppression in subconvulsant doses.

Increase in serum K^{+} concentration following a single dose of suxamethonium is well documented [27, 28]. Tubocurarine has been reported to halve the increase in serum K^{+} [29, 30], although some consider it to be ineffective [28]. In our study, the pretreatment, with both tubocurarine and phenytoin produced a slight but insignificant decrease in mean serum K^{+} levels. Phenytoin may have a membrane stabilising action and delay activation of outward potassium current during the action potentials [18, 31].

Table 4. Characteristics of fasciculations.

Group	No. of patients with fasciculations			Graded intensity of fasciculations Mean (SD)
	Mild	Moderate	Severe	
1	14 (73.7%)	3 (15.8%)	2 (10.5%)	1.37 (0.67)
2	4 (100%)	—	—	1.00 (0.00)
3	8 (42.1%)	10 (52.6)	1 (5.3%)	1.63 (0.58)

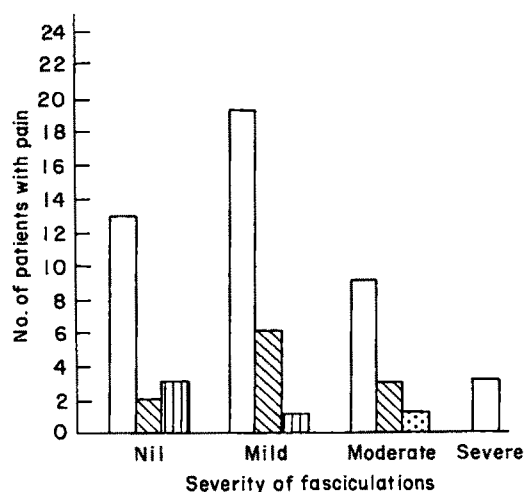


Fig. 3. Relationship between the severity of fasciculations and severity of postoperative muscle pain. □, no pain; ▨, mild pain; ▩, moderate pain; ▤, severe pain.

We observed a significant decrease in serum Na^+ levels after phenytoin administration, an incidental finding. No studies are available on the effect of phenytoin on serum sodium. We have not been able to explain the mechanism of the observed result. It is difficult to say if this effect is entirely due to phenytoin or because of interaction between it and suxamethonium. In view of the fact that a definite decrease in mean serum Na^+ levels was observed in all patients who complained of myalgia, we assume that this may be related to suxamethonium effect on muscles rather than a side effect of phenytoin treatment. This, however, shall need a further investigation.

Our study confirms the findings of others [32, 33], that no simple correlation exists between the severity of muscle fasciculations, serum K^+ changes and the development of postoperative myalgia.

On the basis of this study, we conclude that phenytoin, in doses of 5 mg.kg^{-1} given 5 min before suxamethonium, is effective to prevent suxamethonium-induced myalgia and is superior to tubocurarine. It is cardiostable when given slowly in this dose and should be considered as a pretreatment in all patients where suxamethonium is being used, especially those having day care surgery. It should also be beneficial in rapid sequence intubation as it has the potential advantage of quickening and enhancing suxamethonium-induced neuromuscular block.

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A comparison of laryngeal mask airway with tracheal tube for intra-ocular ophthalmic surgery

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Summary

The use of the laryngeal mask was compared with tracheal intubation in 30 patients who underwent intra-ocular ophthalmic surgery and who received intravenous anaesthesia with propofol. Changes in intra-ocular pressure, heart rate and mean arterial pressure after the insertion of the laryngeal mask airway or the tracheal tube were not significantly different. However, at the end of the procedure, a significantly higher percentage of patients with a tracheal tube coughed, reacted to head movement and suffered breath-holding. In addition, significantly more patients in this group complained of a sore throat ($p < 0.05$). During intravenous propofol anaesthesia, the laryngeal mask airway does not offer any advantage over tracheal intubation in the control of intra-ocular pressure for intra-ocular ophthalmic surgery. However, there were fewer complications immediately following surgery in the laryngeal mask group.

Key words

*Equipment; laryngeal mask, tracheal tube.
Surgery; ophthalmic.*

Control of intra-ocular pressure during intra-ocular ophthalmic surgery is of prime importance. Tracheal intubation is associated with a pressor response, straining, coughing, breath-holding and increases in intra-ocular pressure. The use of the Brain laryngeal mask causes less coughing, straining, breath-holding and an attenuated pressor response [1, 2], and is well tolerated by patients during light anaesthesia and recovery.

The aim of the present study was to assess the use of the Brain laryngeal mask airway and compare it with tracheal intubation in patients who underwent intra-ocular ophthalmic surgery.

Patients and methods

Thirty patients of ASA grade 1, 2 or 3 scheduled to undergo intra-ocular ophthalmic surgery under general anaesthesia were studied after obtaining informed consent. Two patients underwent corneal grafting; all others had cataract extraction with lens implantation. The study was approved by the hospital ethics committee. Thirty patients were allocated randomly to one of two groups with equal numbers in each group. The laryngeal mask airway was

used in group 1 while tracheal intubation was undertaken in group 2. The number of attempts required for the correct placement of the tracheal tube or the laryngeal mask airway was noted. Patients with raised intra-ocular pressure were not studied. All patients received pre-medication with temazepam 20 mg 90 min before operation. On arrival in the anaesthetic room, monitoring was instituted. The electrocardiogram was monitored continuously and arterial pressure was measured at 1 min intervals using a Datascope noninvasive arterial pressure monitor. When the laryngeal mask or tracheal tube was in place, end-tidal carbon dioxide tension ($PE'CO_2$) was measured continuously using an Engstrom capnograph. Intra-ocular pressure was measured in the nonoperated eye by the ophthalmic surgeon, using a Perkin's hand-held applanation tonometer, after instillation of amethocaine and application of fluorescein to the cornea from an impregnated strip. Baseline measurement of intra-ocular pressure was made when the patient arrived in the anaesthetic room. Anaesthesia was induced with propofol using a computerised infusion system [3]. Intra-ocular pressure was measured immediately after induction of anaesthesia, and vecuronium 0.1 mg.kg^{-1} was administered. Intra-ocular

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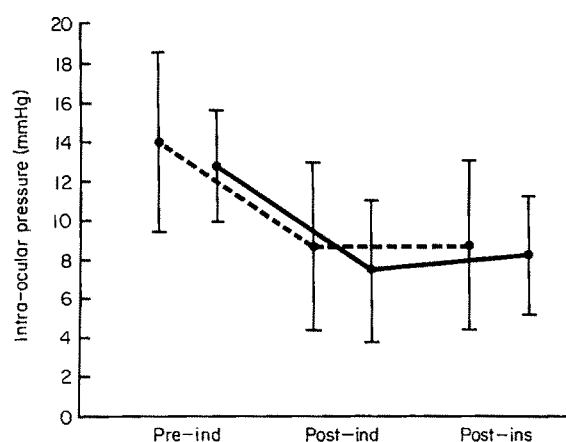


Fig. 1. Intra-ocular pressure before induction of anaesthesia (pre-ind), after induction of anaesthesia (post-ind) and after insertion (post-ins) of either a laryngeal mask airway (●—●) or a tracheal tube (●- -●). Bars represent SD.

pressure was measured again immediately after insertion of a laryngeal mask or tracheal intubation. Controlled mechanical ventilation was carried out to maintain a $PE'CO_2$ of 4.5 kPa during surgery. Anaesthesia was maintained with the propofol infusion system and 60% nitrous oxide in oxygen. Infusion of propofol was stopped on completion of surgery and neuromuscular blockade was reversed using neostigmine 0.35 mg.kg^{-1} given with glycopyrronium 0.07 mg.kg^{-1} . An eye bandage was applied by the surgeon and extubation or removal of the laryngeal mask was carried out after satisfactory establishment of spontaneous breathing. The development of coughing, straining or breath-holding at the termination of anaesthesia was recorded.

Student's *t*-test and Chi-squared test were used for the statistical analysis of the data.

Results

There was no significant difference in demographic data, ASA class, or duration of anaesthesia between the groups (Table 1). No difficulty was encountered in the placement of the tracheal tube, but five patients required more than one attempt to place the laryngeal mask correctly. Because of technical difficulties, it was not possible to measure intra-ocular pressure in one patient in group 1. The mean (SD) peak inflation pressures required for controlled mechanical ventilation of the lungs were $14.5 (4.1) \text{ cmH}_2\text{O}$ in group 1 and $16.0 (3.2) \text{ cmH}_2\text{O}$ in group 2; the difference was not statistically significant.

Table 1. Demographic and anaesthetic data, presented as mean (SD).

	Laryngeal mask (n = 15)	Tracheal tube (n = 15)
Age	6.4 (15)	61 (20)
Males/females	6/9	5/10
Duration of anaesthesia; min	46.6 (21.4)	49.0 (14.0)
Target blood concentration of propofol at the end of operation; $\mu\text{g.ml}^{-1}$	4.1 (0.8)	4.0 (1.3)

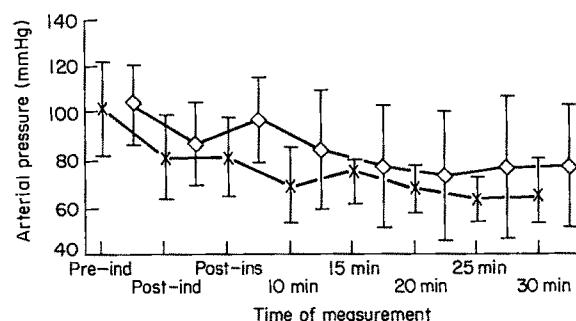


Fig. 2. Mean arterial pressure before induction of anaesthesia (pre-ind), after induction of anaesthesia (post-ind), after insertion (post-ins) of either a laryngeal mask airway (x—x) or tracheal tube (◇—◇), and at 5-min intervals during surgery. Bars represent SD.

Intra-ocular pressures in groups 1 and 2 were 14.0 (4.6) mmHg and 12.7 (2.8) mmHg before induction of anaesthesia, 8.6 (4.2) mmHg and 7.4 (3.4) mmHg immediately after induction and 8.6 (4.4) mmHg and 8.1 (3.0) mmHg 1 min after insertion of the laryngeal mask or tracheal tube respectively (Fig. 1). There were no significant differences between or within the groups in respect of intra-ocular pressure measured before induction, immediately after induction and immediately after the insertion of the laryngeal mask or tracheal tube.

The mean arterial pressure changed from 81.0 (15.0) mmHg to 81.5 (16.6) mmHg on the placement of laryngeal mask and from 87.1 (17.5) mmHg to 97.4 (17.9) mmHg on tracheal intubation (Fig. 2). Heart rate increased from 82.3 (24.2) beat.min^{-1} to 88.3 (27.4) beat.min^{-1} after tracheal intubation; there was a decrease from 76.4 (10.7) beat.min^{-1} to 75.0 (11.9) beat.min^{-1} after insertion of the laryngeal mask (Fig. 3). The changes in mean arterial pressure and heart rate on induction of anaesthesia or the insertion of the tracheal tube or laryngeal mask were not statistically significant.

Computer-predicted mean blood concentrations of propofol at the end of the operation were $4.1 \mu\text{g.ml}^{-1}$ in group 1 and $4.0 \mu\text{g.ml}^{-1}$ in group 2; the difference was not statistically significant. No patient in the laryngeal mask group reacted to movement when the head was lifted for bandaging at the end of operation, while 73% of patients in group 2 strained or coughed ($p < 0.05$; Table 2). Eighty-seven per cent of the intubated patients coughed and 47% held their breath at extubation while no patients coughed or held their breath in group 1 ($p < 0.05$). Postoperative sore throat was reported by 33% of the patients in group 2, while no patient in group 1 complained of sore throat ($p < 0.02$).

Table 2. Frequency of postoperative complications.

	Laryngeal mask (n = 15)	Tracheal tube (n = 15)
Reaction to head movements	0	11*
Cough	0	13*
Breath-holding	0	7*
Sore throat	0	5**

* $p < 0.02$.

** $p < 0.03$.

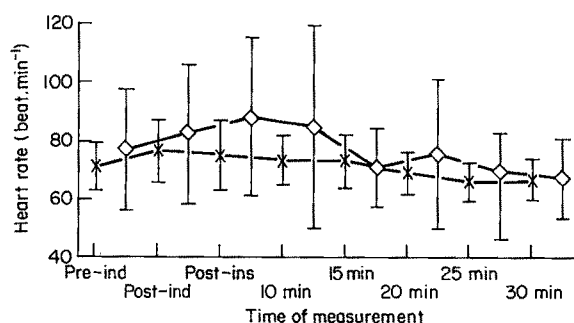


Fig. 3. Heart rate before induction of anaesthesia (pre-ind), after induction of anaesthesia (post-ind), after insertion (post-ins) of a laryngeal mask airway (x—x) or tracheal tube (◇—◇) and at 5-min intervals during surgery. Bars represent SD.

Discussion

The demand for operations such as cataract extraction is increasing with the number of elderly people in the community. Postoperative pain after cataract extraction is minimal and postoperative bleeding is not expected. These characteristics make cataract extraction a suitable day-case operation which may be carried out under local anaesthesia without increase in peri-operative morbidity [4], but for some patients general anaesthesia may be more appropriate and they should not be denied the choice.

An excessive increase in intra-ocular pressure during intra-ocular ophthalmic surgery should be avoided; various factors which affect intra-ocular pressure have been discussed by Holloway [5]. Tracheal intubation is associated with a marked pressor response and increases in intra-ocular pressure [6]. The insertion of a laryngeal mask causes an attenuated pressor response [2] and less of an increase in intra-ocular pressure. In our study, intra-ocular pressure did not change significantly following the insertion of either the laryngeal mask or the tracheal tube. Holden *et al.* [7] reported a significantly greater increase in intra-ocular pressure after tracheal intubation than after insertion of the laryngeal mask. In that study, the larynx was sprayed with 4% lignocaine before tracheal intubation. We did not use lignocaine spray to the larynx and still found no significant difference in intra-ocular pressure between the two groups ($p < 0.05$). We believe that the attenuated increase in the intra-ocular pressure in the present study was due to the use of anaesthesia with propofol. Intravenous propofol anaesthesia has been shown to cause a 30% decrease in intra-ocular pressure from baseline [8] and while the stimulus of tracheal intubation increases intra-ocular pressure, this remains below the baseline level [9].

The peak increase in intra-ocular pressure occurs 20 s after the placement of an airway [10]. We measured intra-ocular pressure immediately after insertion of the tracheal tube or the laryngeal mask, and it is unlikely that the peak increase in intra-ocular pressure was missed.

We did not experience any difficulty in carrying out controlled mechanical ventilation through the laryngeal mask. Successful use of the laryngeal mask airway for intermittent positive pressure ventilation has also been reported by Brain [6]. Although the laryngeal mask has been used successfully for head and neck surgery [11, 12], there remains the possibility that it may be displaced during

surgery with subsequent difficulty in maintaining a clear airway. No displacement of the laryngeal mask occurred in any of the patients we studied, but clinical experience in the use of the laryngeal mask for such procedures is limited. A modification of the laryngeal mask with an armoured tube to prevent kinking has been described for use in otolaryngological and dental anaesthesia [13], and it may also be useful in ophthalmic anaesthesia.

Coughing may increase intra-ocular pressure to more than 50 mmHg [7] and should be avoided, particularly in patients with a perforating eye injury and immediately after intra-ocular ophthalmic surgery. The frequency of coughing at extubation was high in group 2 (87%), while no patient coughed at any time under anaesthesia or at the removal of the laryngeal mask. Holden *et al.* [7] also reported an increased frequency of coughing during extubation compared with the removal of a laryngeal mask, in spite of the use of topical lignocaine. The target propofol concentrations were not significantly different in the two groups; we believe that the depth of anaesthesia in the two groups was similar, and postoperative complications such as coughing and breath-holding were not due to the anaesthetic technique. Deeper anaesthesia at the time of extubation could have prevented coughing but it would have resulted in delayed recovery and compromised the airway. Postoperative sore throat has been reported in up to 12% of patients following the use of the laryngeal mask [14] but no patient in our study complained of sore throat in the laryngeal mask group, while one third of all patients in group 2 had a sore throat. Decreased postoperative morbidity associated with the use of the laryngeal mask may be of advantage. However, the use of the laryngeal mask is not recommended in patients with a full stomach, a history of reflux or hiatus hernia, or chronic bronchitis, as aspiration of gastric contents remains a hazard [15]. The laryngeal mask is not suitable for controlled mechanical ventilation of the lungs in patients with decreased lung compliance or increased airway resistance as this may lead to leakage of the inspired gas, and gastric distension.

During intravenous propofol anaesthesia, the laryngeal mask airway does not appear to offer any advantage over tracheal intubation in the control of intraocular pressure but it decreases significantly the frequencies of postoperative coughing, straining, breath-holding and sore throat.

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A multiple dose comparison of combinations of ibuprofen and codeine and paracetamol, codeine and caffeine after third molar surgery

H. J. MCQUAY, D. CARROLL, P. GUEST, R. P. JUNIPER AND R. A. MOORE

Summary

In a randomised, double-blind, double-dummy, multiple dose, crossover study in 30 patients we compared an ibuprofen/codeine combination (400 mg ibuprofen/25.6 mg codeine phosphate) with a paracetamol/codeine/caffeine combination (1 g paracetamol/16 mg codeine phosphate/60 mg caffeine) for pain relief over 6 days after two-stage bilateral lower third molar removal. The ibuprofen combination produced significantly greater analgesia than the paracetamol combination, both on single-dose analysis of the first and second days and on multiple-dose measures for days 1, 2, 3 and 4. The mean incidence of adverse effects over the 6 days was 20% for both combinations. This trial design (crossover with multiple dosing in outpatients) is a sensitive way of testing for analgesia, and is potentially more predictive of adverse effect problems than single-dose studies. It confirms that multiple dosing may show increased efficacy.

Key words

Analgesics; codeine, ibuprofen, paracetamol.

Pain; postoperative.

Surgery; dental.

Single dose analgesic studies can be sensitive and precise. However, extending these methods to the context of multiple dosing may provide more clinically relevant data [1]. Additional information about the optimal dosing schedule may be gleaned from multiple dosing studies because some analgesics show increased effect on multiple dosing [2] and multiple dosing studies may provide a better evaluation of adverse effects than single doses [3].

Our current postoperative oral surgery model [2, 4] has been shown to be appropriate for testing analgesics in multiple dosing studies. The fact that the patient has the same operation twice (the extraction of a molar tooth on each side), provides ideal circumstances for a crossover design. This design was sufficiently sensitive to show the additional analgesic effect of adding 20 mg of codeine base to 400 mg of ibuprofen compared with 400 mg of ibuprofen alone on both single and multiple dosing [2], although half that dose of codeine did not produce measurable additional benefit [4].

In the present study we compared an ibuprofen–codeine combination with a commercially available paracetamol–

codeine–caffeine combination, developing the multiple dosing aspect by asking patients to record pain measurements throughout the day of surgery and the following days. In addition to standard efficacy measures, a pain intensity and pain relief index per dose was derived over the study period.

Methods

Trial design

Ethics committee approval was obtained for the study, which was of randomised, double-blind, double-dummy, multiple-dose, crossover design (Fig. 1). Informed consent was obtained from 30 patients undergoing elective outpatient surgery for surgical removal of bilateral lower (or lower plus upper) third molars, at the Oral Surgery Department, John Radcliffe Hospital, Oxford. Removing upper third molars, either because they have erupted or are just beneath the mucosa, produces minor and insignificant discomfort when the local anaesthetic wears off compared

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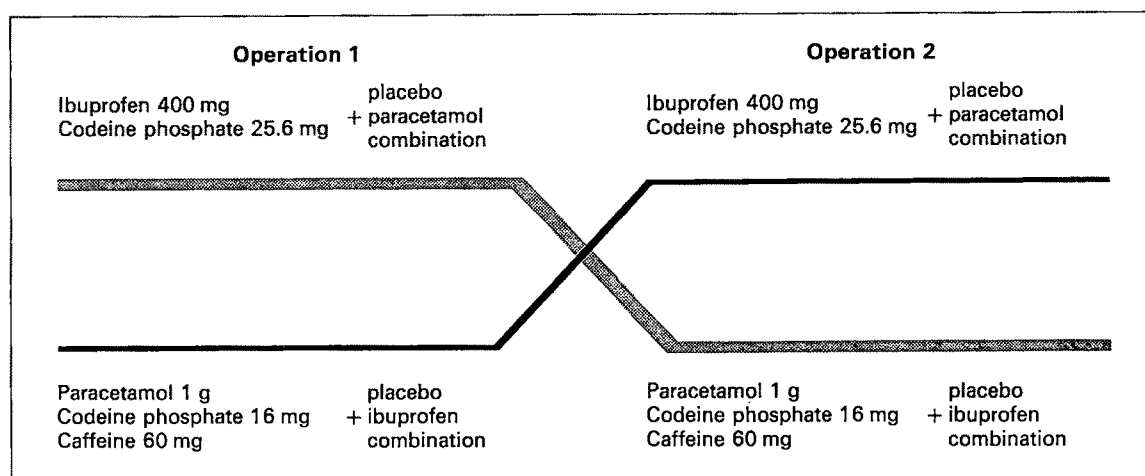


Fig. 1. Study design (10 mg of codeine base is equivalent to 12.8 mg of codeine phosphate).

with removal of lower third molars. After the first operation (removal of the lower third molar on one side), if the patients had pain of moderate or severe intensity they took the first test analgesic and continued to use it for up to 6 days if necessary, at a rate of up to four times a day if required. Diary measurements of pain and pain relief were made on the day of surgery (day 1) and over the next 5 days. The patients were seen 7 days after operation. After a minimum period of 3 weeks they had the second operation and took the second treatment in the same way as on the first occasion.

Test analgesics

The dose of the ibuprofen combination was two tablets, each containing 200 mg of ibuprofen with 12.8 mg of codeine phosphate. The dose of the paracetamol combination was two effervescent tablets, each containing 500 mg of paracetamol with 8 mg of codeine phosphate and 30 mg of caffeine. The trial design was double-dummy with appropriate placebos (Fig. 1). The effervescent placebo tablets were formulated so that there was minimal effect on ibuprofen dissolution. For each dose patients were told to swallow the tablets (envelope 'a') with the dissolved contents of the sachet (envelope 'b'). Envelope 'a' therefore contained two oblong white tablets, either placebo or ibuprofen combination, and envelope 'b' either placebo or paracetamol combination. The fourth dose of the ibuprofen combination for each day was a placebo; this was necessary because of safe prescribing recommendations for the maximum daily ibuprofen dose in this context. The tablet schedules were presented in the diaries (each day's four doses on a separate page). Tablets were identifiable only by patient and treatment numbers. The drugs were supplied by Beecham Products, Weybridge, England.

Patients and operations

We studied men or women aged between 18 and 40 years who were undergoing surgical removal of bilateral symmetrical impacted lower third molars with or without removal of upper third molars. Patients were not studied if they were taking concurrent analgesics, had previously reacted to, or were hypersensitive to, any of the test drugs, or had any gastrointestinal disturbance. Psychiatric

disorders which required medical treatment or any existing illness were grounds for exclusion as were pregnancy or breast-feeding.

Patients were operated on before midday, in order to allow sufficient time for multiple dosing on the day of surgery. Each surgical procedure was carried out under identical conditions, with the same oral surgeon and nursing assistant. The same local anaesthetic (lignocaine 2% with adrenaline 1:80 000) was given before surgery. For each procedure the volume of the local anaesthetic and the time at which it was given were recorded. The type, time and duration of each surgical procedure were recorded. Patients were discharged when the oral surgeon judged that they had recovered fully. Routine post-operative mouth care instructions were given before discharge. Patients were given a prophylactic antibiotic (metronidazole 400 mg) to be taken twice daily for 3 days postoperatively.

Analgesic measurements and adverse effects

Self-report information was obtained in diaries using the following scales: pain intensity, 4-point categorical verbal rating scale (0 = none, 1 = slight, 2 = moderate, 3 = severe); pain relief, a 5-point categorical verbal rating scale (0 = none, 1 = a little, 2 = some, 3 = a lot, 4 = complete); preference, at the postoperative visit 1 week after the second operation the patients were asked to record which of the two treatments they preferred; pain-free hours, the numbers of hours on the day of surgery for which verbal categorical pain intensity scores of zero were noted and were compared for the two treatments, as were the numbers of hours for which maximal verbal categorical pain relief scores were recorded; daily global rating scores, daily global rating scores for the 6 days of each treatment were recorded (0 = poor, 1 = fair, 2 = good, 3 = very good, 4 = excellent).

Adverse effects

Adverse effects were recorded by the patient daily, in answer to the diary question 'Did the tablets upset you in any way?' If the answer was 'yes' the patients were asked to provide details. Any comments made by the patient were discussed at the next clinic visit.

Timing of recordings

The first 2 days. Pain intensity and the time were noted immediately before taking the first dose. The patients then recorded pain intensity and pain relief half an hour after the dose and then hourly until going to sleep.

Days 3, 4, 5 and 6. Daily global rating and timed tablet consumption on days 3, 4, 5 and 6 were noted, together with adverse effects. Patients were seen in the Oral Surgery department 7 days after each extraction, to check that there were no postoperative complications. The diaries and any remaining tablets were then collected, a tablet count was made, and after the second operation the patient's preference was recorded.

Analysis and statistics

Only patients with initial (pre-dose) pain intensity values of moderate or severe on the categorical verbal scale were included in the analgesic measures analysis for the relevant day. This judgment was made separately for day 1 and day 2.

Single dose analysis: Spid and Totpar. Summed pain intensity difference (Spid) and total pain relief (Totpar) [5] were calculated for the 6-h period after the first dose on both the day of surgery and the following day, using the verbal categorical pain intensity and relief scores, respectively. If the patients took another dose before the end of the 6-h study period, then for each reading between the time of remedication and 6 h from the dose, the initial pain intensity reading and a pain relief score of zero were used for the analysis [6].

Repeat dose analysis: PRIX, PRIX/dose, TOTPRIX and TOTPRIX/dose. The pain intensity reduction index (PRIX) [7] was calculated from the categorical verbal pain intensity scale recordings for the first and second days, respectively, using the formula:

$$\text{PRIX} = \frac{\text{initial pain intensity} - \text{mean hourly pain intensity}}{\text{initial pain intensity}} \times \text{no. of hours} \times 100$$

where initial pain intensity was the pain intensity (4-point verbal categorical scale) recorded before the analgesic was taken, and no. of hours was the time from the first dose to the last recorded pain measurement on that day. PRIX/dose was calculated by dividing the PRIX score by the total number of doses of active drug taken on that day. TOTPRIX was calculated from the categorical verbal pain relief scale recordings for the first and second days, respectively, using the formula:

$$\text{TOTPRIX} = \text{mean pain relief} \times \text{no. of hours} \times 100$$

where the no. of hours was the time from the first dose to the last recorded pain measurement on that day. TOTPRIX/dose was calculated by dividing the TOTPRIX by the total of active doses taken on that day.

Statistics

The Wilcoxon signed-ranks matched pairs test was used for a within-patient comparison of analgesic effect scores after the two treatments. Patient preference and adverse effect incidence were compared using the Chi-squared test. The

Table 1. Patient details. Values are expressed as mean (SEM).

	Ibuprofen combination	Paracetamol combination
Number	30	
Age; years	24 (1)	
Sex; M : F	11 : 19	
Weight; kg	66 (2)	
Height; cm	169 (2)	
Duration of surgery; min	10 (1.0)	10 (0.8)
Volume of local anaesthetic; ml	6.4 (0.3)	6.5 (0.3)
Time from local anaesthetic to operation start; min	13 (0.7)	14 (0.7)
Operation type: (lower + upper) vs lower alone	16 : 14	14 : 16
Drill used during surgery	22	22

data are presented as mean (SEM). Significance was assumed at the $p < 0.05$ level.

Results

Details of the patients and of the surgical procedures are shown in Table 1. There were no significant differences between the treatments for the type of operation (lower versus lower and upper third molars), volume of local anaesthetic, duration of surgery or the interval between the local anaesthetic and the start of surgery. More women than men took part in the study. There were no withdrawals from the study because of adverse effects.

Analgesia

Single dose: first day. The ibuprofen combination produced significantly greater Spid ($p = 0.022$) and Totpar ($p < 0.001$) values than the paracetamol combination (Table 2).

Repeated doses: first day. The ibuprofen combination had a significantly greater effect on PRIX ($p < 0.014$), PRIX/dose ($p < 0.01$), TOTPRIX ($p < 0.001$), and TOTPRIX/dose ($p < 0.002$) (Table 2). The mean pain relief scores derived from the verbal pain relief scores at each time point are shown in Figure 2. On day 1 the ibuprofen combination produced a mean TOTPRIX/dose 50% greater than the paracetamol combination.

Single dose: second day. There was no significant difference on either Spid or Totpar (Table 2).

Repeated doses: second day. The ibuprofen combination had significantly greater efficacy only on TOTPRIX/dose ($p = 0.013$; Table 2). The mean pain relief scores derived from the verbal pain relief scores at each time point are shown in Figure 2. On day 2 the mean TOTPRIX/dose was 72% more with the ibuprofen combination than with the paracetamol combination.

Doses taken on days 1 and 2 and dose intervals. The interval between first and second doses on the first day was significantly longer for the ibuprofen combination ($p = 0.001$, Table 2). Over half the patients in each group needed a third dose on that day, with a significantly longer interval between the second and third doses with the ibuprofen combination ($p = 0.023$, Table 2). Five patients took a fourth dose of the ibuprofen combination compared with 12 on the paracetamol combination, and overall for the first day significantly fewer doses were taken with the ibuprofen combination ($p = 0.013$, Table 2). On the second

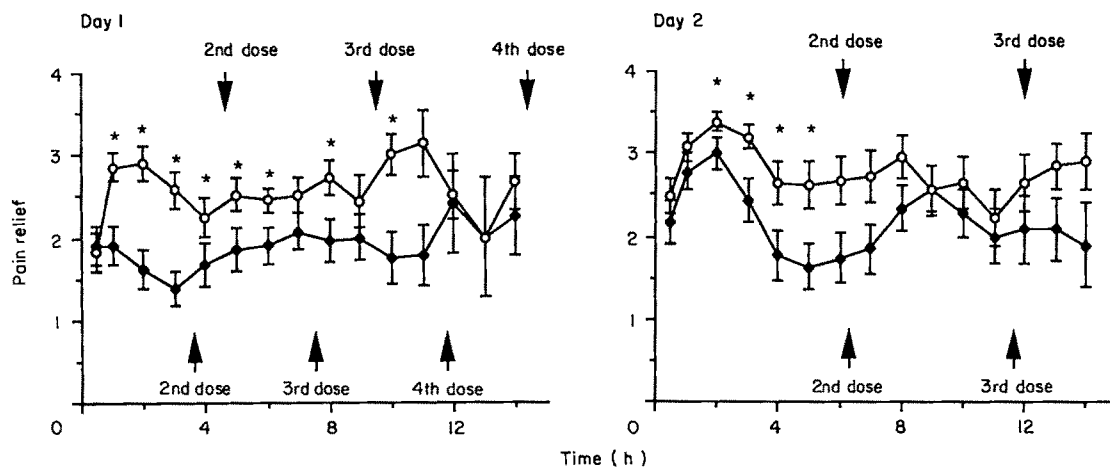


Fig. 2. Mean (SEM) pain relief scores against time for days 1 and 2. —○— ibuprofen combination, —●— paracetamol combination. * Significant difference on Wilcoxon matched-pairs signed-ranks test ($p < 0.05$).

day there were no significant differences in the intervals between doses or in total doses taken.

Patient preference. Twenty-nine preferences were obtained: 21 patients preferred the ibuprofen combination, 8 preferred the paracetamol combination, and 1 expressed no preference ($p = 0.01$, Chi-squared).

Pain-free hours. The ibuprofen combination gave significantly more hours of maximal pain relief and hours of zero pain intensity on the day of surgery than the paracetamol combination (0.5(0.1) vs 0.2(0.1) h, $p < 0.01$ and 0.4(0.1) vs 0.2(0.1) h, $p < 0.1$, respectively). There were no significant differences on the second day.

Daily global rating. There was no significant difference

between the global rating for the two combinations on day 1, but on day 2 the ibuprofen combination gave a significantly higher rating (Table 2).

Efficacy on days 3, 4, 5 and 6. Daily global rating scores and mean doses taken for days 3, 4, 5 and 6 are shown in Table 3. The ibuprofen combination produced significantly better global scores on days 3 and 4.

Adverse effects

The incidence of adverse effects for the two test drugs is shown in Table 4. The incidence of nausea was significantly

Table 2. Analgesic measures days 1 and 2. Values are expressed as mean (SEM).

	Ibuprofen combination (n)		Paracetamol combination (n)		Statistical significance
Day 1					
Pain intensity before first dose	2.2 (0.1)	29	2.2 (0.1)	27	
SPID	3.9 (0.6)	29	2.0 (0.5)	27	0.022
PRIX	406 (56)	29	185 (67)	27	0.014
PRIX per dose	173 (26)	29	93 (33)	27	0.01
Global	2.3 (0.2)	29	1.7 (0.2)	27	
Time to 2nd dose; min	292 (20)	29	217 (15)	26	< 0.001
Time to 3rd dose; min	285 (14)	17	237 (14)	21	0.023
Time to 4th dose; min	287 (93)	5	261 (17)	12	
Time to last reading; min	612 (23)	29	616 (29)	27	
Total doses taken	2.8 (0.1)	29	3.2 (0.2)	27	0.013
TOTPAR	10 (1)	29	5.2 (0.8)	27	0.001
TOTPRIX	222 (13)	29	159 (16)	27	0.001
TOTPRIX per dose	92 (8)	29	62 (11)	27	0.002
Day 2					
Pain intensity before first dose	2.1 (0.1)	24	2.2 (0.2)	21	
SPID	5.9 (0.6)	24	4.9 (0.6)	21	
PRIX	359 (116)	24	478 (66)	21	
PRIX per dose	195 (54)	24	177 (23)	21	
Global	2.7 (0.2)	24	1.9 (0.3)	21	< 0.01
Time to 2nd dose; min	377 (32)	19	376 (26)	19	
Time to 3rd dose; min	310 (21)	13	322 (23)	14	
Time to 4th dose; min	286 (35)	7	259 (41)	7	
Time to last reading; min	707 (46)	24	670 (43)	21	
Total doses taken	2.6 (0.2)	24	3.0 (0.2)	21	
TOTPAR	11 (1)	24	8.1 (1.2)	21	
TOTPRIX	235 (17)	24	167 (18)	21	
TOTPRIX per dose	120 (16)	24	70 (13)	21	0.013

Only patients with moderate or severe pain before taking first dose included in analysis (see text).

Table 3. Analgesic measures and number of doses. Values are expressed as mean (SEM).

	Ibuprofen combination (n)		Paracetamol combination (n)		Statistical significance
Daily global rating					
Day 3	2.6 (0.2)	23	1.9 (0.2)	21	p < 0.003 p < 0.05
Day 4	2.8 (0.3)	20	1.7 (0.4)	10	
Day 5	2.8 (0.3)	16	1.8 (0.4)	10	
Day 6	2.4 (0.4)	9	1.6 (0.5)	8	
Total doses					
Day 3	2.5 (0.2)	23	2.6 (0.2)	21	
Day 4	2.1 (0.2)	20	2.1 (0.4)	11	
Day 5	2.1 (0.3)	17	2.5 (0.3)	10	
Day 6	2.1 (0.2)	9	1.9 (0.4)	8	

Statistical significance from Wilcoxon's test.

higher with the paracetamol combination ($p < 0.05$, Chi-squared). The incidence of adverse effects tended to increase with duration of exposure, but few patients were still taking medication by the last day. The mean percentage incidence (across days) was 21.5% for the ibuprofen combination and 20.9% for the paracetamol combination, higher than for day 1 alone.

Discussion

These results provide further evidence that the effects of postoperative multiple doses of analgesics can be studied and that differences between medications can be detected with this model.

On both single and multiple dosing the ibuprofen combination produced a statistically significant increase in efficacy over the paracetamol combination. This was apparent on both simple measures, such as hours of maximal pain relief, and on the derived indices. Of the derived indices, those calculated from pain relief measures proved more sensitive than those calculated from the pain intensity measures. The ibuprofen combination gave 50% greater TOTPRIX/dose than the paracetamol combination on day 1 and 70% on day 2. This refutes the idea that pain relief measures are unlikely to work well in multiple dosing [8] because the 'anchor point', against which the patient makes the judgment of relief, is long past.

One argument for multiple dosing studies is that they could detect evidence of increased efficacy on multiple dosing. Codeine particularly has a reputation for such an effect [2]. There was some evidence for an increase in this study; the mean TOTPRIX/dose within medication was 36% higher for the ibuprofen combination on day 2 compared with day 1, and 18% higher for the paracetamol combination. Postoperative pain usually decreases with time, so that this judgment of increased effect should be treated cautiously.

The argument that multiple dosing studies can also provide evidence for deciding on optimal dosing strategies is also supported by these results. With patients deciding when to self-medicate within safe prescribing limits, the results showed a significantly greater duration of the effect of the ibuprofen combination compared with the paracetamol combination, in addition to a greater degree of efficacy.

Table 4. Adverse effects (for all patients).

	Ibuprofen combination	Paracetamol combination
Number of patients	30	30
Postoperative secondary infection	2	1
Number of patients reporting adverse effects	9	15
Number of patients reporting more than one adverse effect	4	5
Nausea	3	10*
Drowsy	2	2
Headache	2	0
Dizziness	1	1
Constipation	1	1
Vomiting	0	4
Abdominal pain	1	1
Faint feeling	1	1
Increased bleeding	1	0
Light-headed	1	0
Number of reports of adverse effects	13	20

* Significant difference (Chi-squared).

This multiple dosing model is very sensitive. We asked patients to take the first dose of analgesic when they had moderate or severe pain, rather than to take the drugs prophylactically. The analysis of days 1 and 2 excluded those patients who did not fulfil this criterion; sensitivity might have been lost if this had not been done. An example of this problem is a recent oral surgery comparison of ibuprofen Continus (2×300 mg, $n = 49$) with ibuprofen Continus plus codeine ($2 \times 300 + 20$ mg, $n = 48$) [9]. Prophylactic dosing was used in that study, which may explain the poor sensitivity.

A second general methodological point is the ability to distinguish between true-negative and false-negative results in clinical trials which compare only two treatments and show no difference between them. This particular multiple dosing model can only compare two medications within patients (since lower third molars only occur on two sides). While the present study did show a significant difference between the two treatments, circumstances may arise when this is not the case [9]. More sophisticated design measures may be necessary to guard against this problem.

The clinical relevance of this study is that it showed that an ibuprofen-codeine combination produced better analgesia than a paracetamol-codeine-caffeine combination. Used without an opiate, ibuprofen may be a better choice on its own for postoperative pain relief than opiates such as dihydrocodeine [10]. A nonsteroidal anti-inflammatory drug combined with an opiate may do even better [10-13]. This idea of additive analgesia has been questioned recently [9], but historically such additivity has been well shown. Recent postoperative work [14] has indicated that both components are necessary to achieve maximal pain relief. The disadvantage of such combinations is the potential for an increased incidence of adverse effects, particularly from the opiate component [15]; for example, a higher (60 mg) dose of codeine produced significant adverse effects in older patients. This balance of increased efficacy versus increased adverse effects is unresolved; the present and previous [2] studies in this patient group show that an increase in efficacy can be obtained with little increase in the risk of adverse effects.

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Upper respiratory tract infections and general anaesthesia in children

Peri-operative complications and oxygen saturation

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Summary

Conflicting reports regarding the hazards of anaesthesia in children presenting for surgery with an upper respiratory tract infection have appeared in the literature. In the present study 130 children undergoing general anaesthesia with face mask for myringotomy and grommet insertion were graded as having either an acute or recent upper respiratory tract infection or were asymptomatic according to predetermined clinical symptoms and signs. The severity of respiratory and related complications were scored during induction, emergence and recovery. The peripheral oxygen saturation was recorded during induction, emergence, transfer to the recovery ward and in the recovery ward itself. There were no significant differences ($p > 0.05$) in the complication scores between the three groups of children. However, the incidence of hypoxaemia (oxygen saturation $\leq 93\%$) was significantly greater during transfer in the acute infection group ($p = 0.001$) and the recent infection group ($p = 0.02$), as well as during recovery in the acute group ($p = 0.03$) compared with asymptomatic children.

Key words

Anaesthesia; paediatric.

Infection; upper respiratory tract.

Many children scheduled to undergo myringotomy and insertion of grommet for chronic otitis media suffer from frequent upper respiratory symptoms including sneezing, rhinorrhoea, congestion and cough. These symptoms may be related to the underlying chronic pathology or may be indicative of a viral upper respiratory tract infection (URTI). In general, children presenting for elective surgery with URTIs are cancelled until asymptomatic. Normal children may suffer from an average of three to eight URTIs per year [1] and if surgery was delayed for 4 to 6 weeks after each of these episodes there might be only 4 weeks in the year when the child would be considered fit. The practice of postponing surgery for patients presenting with an URTI stems from the widely held belief that anaesthesia increases the risk of peri-operative complications. Although several studies support this viewpoint, others suggest that for certain procedures there is no increased risk for patients presenting with acute, uncomplicated URTIs [2–5]. Part of the difficulty in evaluating and comparing these studies is due to the confounding effect of variations in surgical site, tracheal intubation, patient age, upper respiratory infection criteria and lack of definition or gradation of 'respiratory complications'.

Whereas most investigations of upper respiratory tract infection and anaesthesia have focused on the incidence of rare, major complications such as complete laryngospasm, intra-operative lobar collapse and postoperative pneumonia, the presence of more common respiratory complications, such as partial airway obstruction, excessive secretions, coughing and oxygen desaturation has not been quantified. Postoperative hypoxaemia has been documented in symptomatic and asymptomatic children with pulse oximetry [6, 7] but possible intra-operative hypoxaemia has yet to be evaluated.

The present study was designed to determine if a child with symptoms and signs consistent with an upper respiratory tract infection undergoing mask anaesthesia for myringotomy and insertion of grommet is at increased risk for peri-operative respiratory complications or hypoxaemia.

Methods

This study was approved by the local Ethics Review Committee and informed consent obtained from a parent before anaesthesia. One hundred and thirty children

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Table 1. Scoring system for each of the seven complications: minimum score = 7, maximum score = 28.

	1	2	3	4
1. Cough	None	Occasional	Frequent	Continuous
2. Breath holding	None	< 15 s	15–30 s	> 30 s
3. Airway obstruction (relieving factors)	None	Partial (reposition airway)	Partial (CPAP)	Complete (muscle relaxant)
4. Bronchospasm	None	Expiration only	Inspiration Expiration	Difficult to ventilate
5. Secretions	None	Minimal no suction	Moderate suction × 1	Copious suction > 1
6. Excitement	None	< 30 s	30–60 s	> 60 s
7. Nausea, vomiting or regurgitation	None	Nausea	Retching	Vomiting or regurgitation

conforming to ASA grades 1 or 2, undergoing myringotomy and insertion of grommet were studied. Children were placed into one of three groups based upon their presenting symptoms: group 1, acute URTI present; group 2, URTI within the last 2 weeks but asymptomatic on the day of operation and group 3, no URTI symptoms for more than 2 weeks. Upper respiratory tract infection was defined according to the criteria used by Tait and Knight [8]. The presence of two or more of the following symptoms were required (reported by parent or child): (1) sore or scratchy throat, (2) sneezing, (3) rhinorrhoea, (4) congestion, (5) malaise, (6) nonproductive cough, (7) fever less than 101°F or (8) laryngitis. Combinations of (1) and (5), (2) and (3), (3) and (6), or (4) and (6) required the presence of one additional symptom. Children with a fever greater than 101°F, productive cough or evidence of a lower respiratory tract infection were not studied.

The children received no premedication and a baseline oxygen saturation was obtained before induction of anaesthesia with the patient breathing room air. Anaesthesia was induced with 66% nitrous oxide in oxygen followed by 1–3% halothane by face mask and Mapleson D system. During surgery all children continued to breathe spontaneously the same concentrations of nitrous oxide and oxygen, with up to 2% halothane. At the conclusion of the procedure 100% oxygen was administered for 3 min following the discontinuation of the anaesthetic agent and immediately before transfer to the recovery ward. The electrocardiogram, blood pressure (Dinamap) and peripheral oxygen saturation (Nellcor N-100 pulse oximeter) were monitored during induction, maintenance and emergence from anaesthesia.

A blinded observer scored the induction, emergence and recovery periods for the incidence and severity of the following complications: (1) airway secretions, (2) coughing, (3) breath holding, (4) bronchospasm, (5) upper airway obstruction including laryngospasm, (6) excitement or restlessness and (7) vomiting or regurgitation. These complications were each rated on a four-point scale and

then pooled to obtain a total score. For example, airway obstruction was rated as either (1) absent, (2) partial, (3) partial, requiring positive pressure ventilation to overcome or (4) total, requiring muscle relaxation with or without intubation. The total complication score could therefore range from a minimum of 7 to a maximum of 28 (Table 1). Oxygen saturation was recorded by the same blinded observer every minute during induction, emergence and transport to the recovery room, and was subsequently recorded every minute for the first 5 min in the recovery room followed by every 5 min for the next 15 min. Supplemental oxygen was given immediately in the recovery room if the oxygen saturation fell below 90%. The duration of surgery, anaesthesia, transfer from the operating room to the recovery room and time to discharge (taken from the end of anaesthesia to discharge from hospital) were also recorded.

Continuous data were analysed using analysis of variance (ANOVA). Analysis of categorical variables was performed using a contingency table (expanded Chi-square) and the Bonferroni correction was applied when multiple group comparisons were made. A *p* value of < 0.05 was considered statistically significant.

Results

One hundred and thirty children were studied; 22 fulfilled the criteria for the acute URTI group, 28 the recent URTI group and 80 the asymptomatic group. There were no statistically significant differences in age or sex distribution between the acute infection, recent infection or asymptomatic groups (Table 2). There were also no differences between the groups in terms of the duration of surgery, anaesthesia and transfer or the time to discharge (Table 2). None of the children received atropine.

There were no significant differences in the complication scores during induction, emergence or at any time during the recovery period before discharge from hospital (Table 3). There were also no significant differences in the

Table 2. Demographic and perioperative data. Mean (SD or %).

	Acute URTI	Recent URTI	Asymptomatic
Age; months	47.2 (44.7)	29.0 (26.4)	46.1 (40.3)
Weight; kg	17.7 (10.0)	13.3 (6.1)	16.9 (9.4)
Sex (M)	15 (68)	19 (68)	51 (64)
(F)	7 (32)	9 (32)	29 (36)
Duration of surgery; min	8.6 (3.9)	8.9 (3.5)	9.7 (4.4)
Duration of anaesthesia; min	15.0 (5.3)	15.3 (5.0)	16.2 (5.5)
Duration of transport; s	88.0 (26.0)	95.0 (33.0)	89.0 (41.0)
Time to discharge; min	65.9 (14.3)	66.2 (13.4)	66.5 (17.6)

Table 3. Complication scores at induction, emergence and recovery. Mean (range).

	Acute URTI (n = 22)	Recent URTI (n = 28)	Asymptomatic (n = 80)	p
Induction	7.9 (7-15)	8.5 (7-12)	8.0 (7-12)	0.16
Emergence	7.6 (7-10)	7.6 (7-12)	7.6 (7-12)	0.52
Recovery	8.8 (7-15)	8.7 (7-12)	8.6 (7-15)	0.91

Table 4. Number (%) of children with scores of seven (no complication) at induction, emergence or recovery.

	Acute URTI (n = 22)	Recent URTI (n = 28)	Asymptomatic (n = 80)	p
Induction	12 (55)	10 (36)	36 (45)	0.41
Emergence	13 (59)	16 (57)	62 (77)	0.06
Recovery	7 (14)	10 (20)	32 (65)	0.76

incidence of children with scores of 7 (no complication) at induction, emergence or recovery (Table 4). Total laryngospasm requiring intubation or muscle relaxants did not occur. One child in the asymptomatic group developed laryngospasm which was successfully treated with CPAP (continuous positive airway pressure) applied by means of the mask. Mild, partial laryngospasm occurred in 10 patients during induction (four with acute infection, one with recent infection and five asymptomatic cases) three patients during emergence (all in the asymptomatic group) and three patients in the immediate recovery period (all in the asymptomatic group). None of these findings were statistically significant.

The oxygen saturation did not differ significantly between groups before or during induction of anaesthesia (Table 5). However, there were significant differences between the three groups during transfer and recovery (Table 5). The incidence of hypoxaemia (oxygen saturation $\leq 93\%$) was significantly greater during transfer in the acute infection group ($p = 0.001$) and the recent infection group ($p = 0.02$), as well as during recovery in the acute group ($p = 0.03$) compared with asymptomatic children. Hypoxaemia during transfer did not appear to depend on the duration of transfer or the age of the child. Twenty-one children in our study were under one year of age (15, asymptomatic; four, recent URTI; two, acute URTI) and 35 were over 5 years of age (24, asymptomatic; three, recent URTI; eight, acute URTI); none of these patients had a saturation less than 94% during transfer. We were unable

to record oxygen saturations on six children during transfer due to excessive movement causing interference with the pulse oximeter (one, acute URTI; one, recent URTI; four, asymptomatic) and their data were excluded from analysis (Table 4). There were no complications other than those reported; no child required hospitalization and there were no significant differences in discharge times between the three groups.

Discussion

The management of the patient presenting with an upper respiratory tract infection for elective surgery is a perennial clinical dilemma. Despite the medical, social and financial implications of delaying treatment few studies have addressed this issue. Some of these are anecdotal or poorly controlled and comparisons are hindered by differences in study design.

Part of the controversy surrounding anaesthetic risk and upper respiratory tract infection is due to the problem of defining when such a condition is present. Confirmatory evidence of an URTI should ideally include viral cultures with acute and convalescent antibody titres, but this is clearly neither practical nor cost-effective for a self-limiting, usually benign illness. In a previous study by Tait and Knight [3] nasopharyngeal cultures were taken from a random sample of symptomatic and asymptomatic children. They confirmed the presence of virus in about 25% of cases overall, although this is likely to be an underestimate. Consequently, most papers describing the management of the patient with upper respiratory symptoms must base their definition on the presence or absence of clinical symptoms. Unfortunately, there are no unique clinical criteria for the diagnosis of upper respiratory tract infections and they may be difficult to distinguish from some allergic conditions. The criteria used in our study have been used previously and attempt to identify the types of symptom that are routinely used by clinicians to make a decision regarding the patients' status [8]. These criteria, together with information obtained from the child's parents as to the nature of the symptoms, were used to categorise the patient into one of three groups.

There are few previous investigations designed to determine if children with an upper respiratory tract infection are at increased risk of peri-operative respiratory complications. In 1979, McGill *et al.* [2] reported 11 cases of pulmonary dysfunction following general anaesthesia. Coarse breath sounds, despite suctioning and radiographic

Table 5. Minimum oxygen saturation during induction, transfer and recovery: incidence (%).

	Acute URTI	Recent URTI	Asymptomatic	p
Induction				
$\geq 94\%$	21 (95)	26 (93)	76 (95)	0.89
$\leq 93\%$	1 (5)	2 (7)	4 (5)	
Transfer				
$\geq 94\%$	15 (71)*	24 (89)†	75 (99)	0.001
$\leq 93\%$	6 (29)	3 (11)	1 (1)	
Recovery				
$\geq 94\%$	15 (68)¶	21 (75)	72 (90)	0.02
$\leq 93\%$	7 (32)	7 (25)	8 (10)	

*Acute vs asymptomatic $p = 0.001$; †recent vs asymptomatic $p = 0.02$; ¶acute vs asymptomatic $p = 0.03$ (contingency table with Bonferroni correction).

evidence of atelectasis or infiltrate were noted in 10 children who gave a history of respiratory infection during the 4 weeks before surgery. A retrospective study by Tait and Knight [8] of 3585 children with acute, recent or no upper respiratory infection undergoing various operative procedures found a small but significant increase in the prevalence of respiratory complications (laryngospasm, bronchospasm, breath-holding, stridor) in the recent infection (within 2 weeks) group. In a follow-up prospective study, the same authors [3] investigated 489 children undergoing myringotomy under general anaesthesia with a face mask. This time there were no significant differences in complications (laryngospasm, bronchospasm, apnoea or arrhythmias) between children with an acute, recent or no infection, although oxygen saturations were not recorded. These latter findings confirm those of the present study. In contrast, however, Cohen and Cameron [4] examined a large paediatric database which was prospectively obtained from 1982 to 1987 and found that children with an upper respiratory infection were 2 to 7 times more likely to experience 'respiratory related adverse events' during the intra-operative, recovery and postoperative phase. Furthermore, these risks were increased by tracheal intubation.

Some of the differences between these studies may be explained by several factors. Firstly, some of the patients in the study by Cohen and Cameron [4], the majority of patients in Tait and Knight's earlier retrospective study [8], and all of the patients in McGill's case reports [2] were intubated for their procedures. Bypassing the nasopharynx and oropharynx with high flows of dry gases encourages the inspissation of secretions leading to mucus plugging and atelectasis. This effect may be more pronounced with longer, major procedures. Tracheal intubation may also lead to, or exacerbate, intraluminal swelling of the trachea which can have significant physiological implications for the paediatric patient [5]. Secondly, the earlier studies by Tait and Knight [4] and McGill *et al.* [2] are retrospective. The study by Cohen and Cameron is strengthened by the size of their database ($n = 29\,220$) but the upper respiratory tract infection was not defined and there was scanty information concerning the timing of the infection relative to the date of the surgical procedure. None of these investigations included data on peripheral oxygen saturation and all lacked a uniform anaesthetic regimen.

One previous study has investigated peri-operative hypoxaemia in children with upper respiratory infections. DeSoto and coworkers [9] found that children with clinical symptoms of infection have a different pattern of oxygen saturation compared to asymptomatic children. These workers found a significantly higher incidence of transient hypoxaemia (oxygen saturation $< 95\%$) in the recovery room in children with signs of respiratory infection at the time of surgery or in the preceding week compared to asymptomatic children.

In the present study we were able to demonstrate that children with an acute upper respiratory tract infection have a higher incidence of hypoxaemia (oxygen saturation $\leq 93\%$) during transfer and recovery. Children who developed hypoxaemia during transfer tended to continue desaturating in the immediate recovery period. During this investigation supplemental oxygen was given only if the peripheral oxygen saturation fell below 90% ; all such children responded with a rapid rise in oxygen saturation.

None of the children in the study by DeSoto and colleagues [9], or the present study, developed post-operative respiratory complications or required a prolonged hospital stay.

In a study of hypoxaemia after general anaesthesia in healthy children, Motoyama and Glazener [7] found the mean peripheral oxygen saturation on arrival in recovery to be 93% (corresponding to a calculated arterial oxygen tension of approximately 66 mmHg). This figure was significantly different from the pre-operative mean of 97.6% . These authors suggested that supplementary oxygen should be given in the postoperative period at least until the child is awake enough to reject it. We chose to define hypoxaemia as $\leq 93\%$ because of the findings by Motoyama and Glazener [7] and because we felt that supplemental oxygen should normally be given at this point before further desaturation (corresponding to the steep part of the oxygen-haemoglobin dissociation curve) occurred.

An editorial in the *British Journal of Anaesthesia* [10] suggested that there was little evidence to show that anaesthesia in adult patients with upper respiratory infection predisposed to respiratory complications. Our data suggest that children with an acute or recent upper respiratory infection have an increased likelihood of transient hypoxaemia in the peri-operative period, which responds promptly to supplemental oxygen therapy. We believe that minor surgery not requiring tracheal intubation can proceed in children who have acute or recent infection and who are not toxic. Children with a fever $> 101^\circ\text{F}$, productive cough or evidence of a lower respiratory tract infection were not studied. We advocate continuous monitoring with pulse oximetry during the peri-operative period and the routine use of supplemental oxygen therapy during both transfer and recovery.

Acknowledgments

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Patient-controlled sedation using propofol in day surgery

P. GRATTIDGE

Summary

Patient-controlled sedation using propofol was studied in 30 unselected patients aged 26–72 years undergoing day case general surgical or orthopaedic procedures under local or regional anaesthesia. Using a fixed propofol dose of 0.7 mg.kg^{-1} body weight, the technique provided anxiolysis and sedation with few side effects and a high degree of patient satisfaction. Increasing age was significantly correlated with a deeper maximum level of sedation ($p < 0.001$), indicating a need for a lower bolus dose in the higher age groups. Interpatient variability in propofol requirement was reflected in the lack of significant correlation between propofol dose and procedure duration ($p = 0.095$) and patient age ($p = 0.72$).

Key words

Anaesthesia; outpatient.

Anaesthetics, intravenous; propofol.

Anaesthetic techniques; patient-controlled sedation.

Surgical procedures carried out under local or regional anaesthesia are associated with patient discomfort and apprehension, the extent of which varies from patient to patient. Patient-controlled sedation (PCS) with propofol has been used successfully during extraction of third molars, allowing the patient to titrate drug dosage on an individual basis [1]. The present study was undertaken to test the feasibility of using a similar method in general surgical and orthopaedic day care patients.

Methods

The study was approved by the Regional Research Ethics Committee and all patients gave informed consent.

Thirty ASA 1–2, nonselected, consecutive, unpremedicated outpatients scheduled for elective general surgical or orthopaedic procedures under local or regional anaesthesia were instructed in the use of an unmodified, commercially available patient controlled analgesia machine (PCAS, Graseby Medical Ltd, Watford, England). Instruction was given orally by the anaesthetist when the patient arrived at the Day Surgery Unit on the day of surgery and was reinforced by a written description to be read by the patient whilst waiting for transfer to the operating room. The pump, charged with propofol 10 mg.ml^{-1} , was programmed to deliver on demand a bolus dose of 0.7 mg.kg^{-1} body weight at a rate of 16.7 ml.min^{-1} with a minimum lockout period of 3 min after each delivered

dose. This dose was chosen as it had proved satisfactory in a previous study of intra-operative PCS [1]. Prior to the anaesthetic block being given, and immediately after pump connection to an intravenous cannula inserted into a peripheral vein, the patient was encouraged to make the first demand by pressing the hand-held triggering device and subsequently to make a demand in the event of any discomfort.

The electrocardiogram (ECG), arterial blood pressure and oxygen saturation were monitored throughout the procedure. All patients breathed air or oxygen-enriched air. Maximum depth of sedation attained during the procedure was rated by the anaesthetist on a five-point sedation scale that extended from 1 = fully awake to 5 = unrousable to mild physical stimulation (Table 1) [2].

At the end of the surgical procedure, the pump was disconnected and the following recordings were read from the PCAS display: total dose of propofol administered; number of attempts at self-administration of propofol and

Table 1. Sedation scores.

1.	Fully awake and orientated
2.	Drowsy
3.	Eyes closed but rousable to command
4.	Eyes closed but rousable to mild physical stimulation (earlobe tug)
5.	Eyes closed but unrousable to mild physical stimulation

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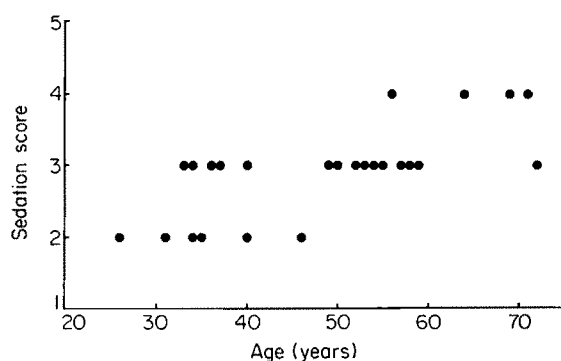


Fig. 1. Relationship between patient age and maximum sedation score, $r = 0.63$, $p < 0.001$ ($n = 30$).

the number of successful attempts. The length of time PCS was available to the patient was taken as 'procedure duration'. Side effects and complications were registered per- and postoperatively. Prior to discharge from the Day Surgery Unit patients were asked their opinion of the peri-operative sedation technique and its effect.

Pearson's correlation coefficient was used to compare the relationship of propofol dose with procedure duration and patient age. Spearman's rank correlation was used for analysis involving sedation score and number of attempts at self-administration. A p value of less than 0.05 was taken to be significant.

Results

Of thirty-one patients approached, one declined to enter the study, preferring that the anaesthetist administer sedation as he saw fit. Of the remaining 30 patients (21 female) the mean age was 46 years (range 26–72) and mean weight 69 kg (range 50–100). The type of anaesthetic block given was intravenous regional in 19 cases, local infiltration in seven cases, and spinal in four cases.

No technical problems were encountered with the PCAS equipment. All patients understood the aim and function of the sedative technique and throughout the procedure were readily able to use the pneumatic triggering device in a purposeful manner, as and when they felt it necessary. There was a smooth onset of sedation, due to an infusion time for each dose of approximately 2.5 min.

The mean total number of attempts at self-administration was 5.0 (SD 3.2). Out of these a mean of 3.8 (1.4) were successful and 1.2 (2.4) were unsuccessful. Thus, most demands fell outside the lockout period and resulted in bolus-dose administration. This indicates that infusion dose and availability were adequate for the patient to reach and maintain the desired sedation target level. The mean procedure time was 48 min (18) and was not significantly correlated with the total number of attempts at self-administration ($r = 0.13$, $p = 0.49$).

The deepest level of sedation reached during the procedure corresponded to full eyelid closure with prompt response to mild physical stimulation and occurred in four patients aged 56, 64, 69 and 71 years respectively. Thus, 26 patients (87%) became drowsy or were so lightly sedated that they could respond to spoken commands at all times. There was a highly significant correlation between sedation score and age ($r = 0.63$, $p < 0.001$) (Fig. 1).

Two male patients (46 and 69 years) showed a transient

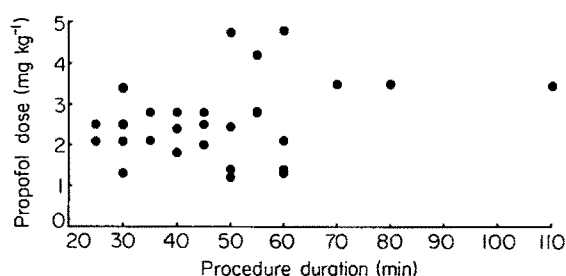


Fig. 2. Relationship between procedure duration and propofol dose, $r = 0.31$, $p = 0.095$ ($n = 30$).

fall in oxygen saturation under 95%, as a result of depressed and obstructed respiration respectively, occurring at the time of peak effect of the bolus dose. A further two patients complained of transient, mild discomfort at the infusion site during the first bolus dose. Cardiac rhythm and rate, together with blood pressure, remained within expected limits for the surgical procedure and anaesthetic block. No changes that occurred could be related to the propofol infusion as such.

The mean total dose of propofol administered was 2.6 mg.kg⁻¹ (0.9). Correlation with procedure duration was weak and did not reach statistical significance ($r = 0.31$, $p = 0.095$) (Fig. 2). This suggests that procedure time alone was not the determining factor for the amount of propofol used. The range of values for propofol requirement was wide (1.2–4.8 mg.kg⁻¹) and they did not correlate significantly with patient age ($r = -0.07$, $p = 0.72$).

All patients were awake on leaving the operating room, and no nausea or vomiting occurred postoperatively. Fluids and analgesics as required could be taken orally almost immediately.

Interviewed prior to leaving for home all patients were satisfied with the sedation itself and its method of delivery. Many described a feeling of well-being and relaxation during the procedure and found the experience of being free to exert a degree of direct control over their treatment to be both novel and beneficial. Some commented that they did not remember much of the procedure and ascribed this to having been asleep part of the time.

Discussion

The trend in health care towards more active patient participation, as exemplified by the concepts of informed consent and patient-controlled analgesia, affects patient attitudes and expectations. Seen in this context, the technique of PCS is a natural complement. The findings of the present study lend support to its feasibility and positive contribution to peri-operative patient care. This view is supported by recent work showing that improved intra-operative comfort during epidural anaesthesia could be related to the positive psychological effect of using a PCS technique [3]. A report on the administration of propofol by patient-controlled infusion in a very small dose (10 mg), as premedication in outpatients, demonstrated the value of this agent as an anxiolytic [4]. Effective anxiolysis, perceived by the patient as being of high quality, was also a finding of the present study.

Except for the initial period, while the patient was being settled and the anaesthetic given, procedures in the study group were associated with a low level of discomfort.

Patients were able to adjust administration of propofol to maintain drowsy anxiolysis or light sleep according to individual preference. This is expressed in the results as an interpatient variability in drug requirement that lacks simple linear correlation with procedure duration. In contrast, in procedures where the level of discomfort is higher, such as during dental surgery, significant correlation could be demonstrated between these variables [1].

With advancing age, sedation dose requirement for propofol falls [2]. In the present study this is reflected in the deeper level of sedation reached by older patients and suggests that a smaller bolus dose than 0.7 mg.kg^{-1} may be more appropriate above a certain age. Further investigation of this aspect of PCS with propofol is required. Another observation of possible relevance was that respiratory complications occurred only in males. However, the number of patients was too small for any conclusions to be drawn.

Propofol and midazolam have been compared when given by continuous infusion during procedures under spinal anaesthesia [5]. Propofol's relative freedom from side effects, together with rapid recovery and a low level of postoperative amnesia were judged to be of significant benefit. Similar properties in favour of propofol were demonstrated in a study of anaesthetist-administered midazolam and fentanyl compared with PCS using propofol [6]. Thus, propofol could be expected to be of particular advantage in day surgery patients and this is

borne out by the findings of the present study. That propofol is sufficient as a single agent, removes the need to use intravenous analgesics with their attendant potential for undesirable side effects. A smooth recovery period that is pleasant to the patient and that does not delay discharge from the Day Surgery Unit is thereby facilitated.

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CASE REPORT

An overdose of ecstasy

A role for dantrolene

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Summary

An overdose of the semisynthetic, hallucinogenic amphetamine 3,4-methylenedioxymethamphetamine resulted in convulsions, hyperthermia, hyperkalaemia and rhabdomyolysis. The patient's management, which included the use of dantrolene, is discussed.

Key words

Toxicity; 3,4-methylenedioxymethamphetamine (MDMA), 'Ecstasy'.

Complications; convulsions, rhabdomyolysis, hyperthermia.

Pharmacology; dantrolene.

The semisynthetic amphetamine 3,4-methylenedioxymethamphetamine (MDMA) also known as 'Ecstasy', has gained popularity in recent years as a recreational drug in the USA and UK. Amphetamine overdose is well known to cause sympathetic stimulation, hyperthermia, convulsions, coma and death. We report a case of a massive overdose of MDMA which was successfully treated with the early use of dantrolene.

Case history

A 24-year-old, otherwise fit male was admitted after having collapsed at a local night club during a so-called 'Rave Party'. Past medical history was unremarkable and he had undergone a previous operation for a splenectomy after a road traffic accident. There was no history of anaesthetic problems noted in either the patient or his immediate family. The patient was reported to have taken 200 mg of MDMA.

On admission he was unconscious, cyanosed and in status epilepticus, which remained refractory to treatment with 30 mg intravenous diazepam. His blood pressure was recorded as 90/60 mmHg with a supraventricular tachycardia of 170 beat.min⁻¹. Axillary temperature was 40.2°C. The convulsions were controlled with thiopentone 400 mg, his trachea was intubated without the use of suxamethonium and intermittent positive pressure ventilation (IPPV) with 100% oxygen was given. A catheter was inserted into the right internal jugular vein.

Initial blood gas analysis after intubation and IPPV were as follows: pH 6.76, P_aO₂ 36.8 kPa, P_aCO₂ 11.9 kPa, bicar-

bonate 12 mmol.l⁻¹ and base excess -12 mmol.l⁻¹. Initial biochemistry showed a hyperkalaemia, with serum potassium 6.6 mmol.l⁻¹, sodium 140 mmol.l⁻¹, urea 5.7 mmol.l⁻¹ and glucose 9.8 mmol.l⁻¹. The only abnormality on the first full blood count was a leukocytosis of 35×10^9 l⁻¹. This was repeated a short while later and was found to be 52.1×10^9 l⁻¹ with a normal differential count. The film showed a left shift with toxic granulation and the first coagulation studies were normal.

The patient was transferred to the intensive care unit (ITU) and IPPV, with a minute volume of 12 l.min⁻¹, an F_{IO}₂ of 0.5 and rate of 15 breath.min⁻¹ was started. Neuromuscular blockade with an atracurium infusion and sedation with midazolam was instituted. The hyperkalaemia proved refractory to treatment; the first infusion of 50 ml 50% glucose with 15 units human actrapid insulin did not result in any significant decrease in serum potassium levels. Only after the third such infusion did we obtain a response, in which the serum potassium decreased from 6.4 to 4.9 mmol.l⁻¹.

The hyperthermia was treated at first with surface cooling, using moistened towels, fanning and body exposure. Intravenous fluids were chilled in a refrigerator before infusion. Five litres of crystalloid (alternating 5% dextrose and dextrose saline) were administered in the first 8 h, to compensate for the profuse sweating and also as prophylaxis against renal failure secondary to rhabdomyolysis and myoglobinuria.

After discussion with staff at the Guy's Hospital Poisons Centre, we instigated dantrolene infusions an hour after admission, at which time the patient's rectal temperature

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was 40°C. The first dose of dantrolene 1 mg.kg⁻¹ failed to reduce the temperature and two further bolus infusions were given, to a total of 3 mg.kg⁻¹ within an hour. The temperature and heart rate then began to decrease, reaching normal levels within a few hours. A maximum serum creatine kinase of 96550 IU.l⁻¹ was noted on day two, decreasing to 48000 IU.l⁻¹ by day three, in parallel with the disappearance of myoglobin from the urine. Arterial blood gases improved steadily and the PaCO₂ and pH reached normal values within 8 h. No bicarbonate was administered.

Intermittent positive pressure ventilation, sedation and neuromuscular blockade were maintained for 48 h and then the patient was weaned from the ventilator without incident. All laboratory investigations returned to normal except for a transient rise in the International Normalized Ratio (INR) to 1.6 at day two. The maximum recorded temperature was 40.2°C axilla (on admission) and the peak serum potassium level was 6.6 mmol.l⁻¹, also on admission. Myoglobinuria, secondary to rhabdomyolysis, resolved within 2 days. The patient remained in hospital for a further 2 weeks awaiting a formal psychiatric assessment, which showed an organic brain syndrome and an underlying personality disorder.

Discussion

MDMA is taken with a view to feeling 'mellow' and contented and is understood, in lay terms at least, to be relatively safe and nonaddictive. It has been promoted as a chemical adjunct to psychotherapy [1] and is a schedule 1 drug in the USA. Its effects [2] include a sensation of closeness, bruxism, trismus, muscle aches, tachycardia, and depression. MDMA and 3,4-methylenedioxymphetamine (MDA) are structurally related to methamphetamine and have sympathomimetic properties. Mescaline, a hallucinogen, is chemically similar [3]. There is evidence for the biotransformation, in man, of MDMA to MDA [4], which has been shown to be neurotoxic and to destroy serotonergic (5-hydroxytryptamine) nerve terminals in the brain [5].

Reports of MDMA overdose are rare. Four cases have been documented in the US medical literature [3, 6]; two deaths probably resulting from cardiac arrhythmias, one fatal accident when an MDMA user jumped from an electrical utility tower and one nonfatal case involving multiple complications, including hyperthermia. One fatality has been reported in the UK which involved coagulopathy and hyperthermia [7]. Rhabdomyolysis was mentioned as a complication in one patient [6], but without further details or biochemical documentation.

Dantrolene was not used in the management of any of the above cases. It has been used in a case of fatal theophylline poisoning [8], but we believe this to be the first reported use of dantrolene in the UK for MDMA overdose. In view of the response to dantrolene in patients with

malignant hyperthermia, it seemed worthwhile to try using it for amphetamine-induced hyperthermia. In this case it reduced the patient's hypermetabolic state, as measured by the decrease in temperature and heart rate which occurred after it had been given. No comment can be made about further convulsions, since no cerebral function monitor was available and the patient's muscles were paralysed. Although it might be argued that supportive treatment alone would have been effective eventually, it was felt that early and aggressive management was appropriate in this patient. Parr *et al.* reach a similar conclusion in their report [8].

Dantrolene, first marketed as a muscle relaxant, acts by inhibiting the release of calcium from the sarcoplasmic reticulum and has been shown to reduce mortality in malignant hyperthermia. It has also been recommended in the treatment of theophylline overdose [8] and, in combination with dopamine agonists such as bromocriptine, it is used in the treatment of the neuroleptic malignant syndrome [9].

In conclusion, we suggest that dantrolene should be used in the management of MDMA overdose whenever hyperthermia supervenes.

Acknowledgments

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CASE REPORT

The laryngeal mask airway

Two cases of prehospital trauma care

M. K. GREENE, R. RODEN AND G. HINCHLEY

Summary

Two cases of emergency prehospital airway control using the laryngeal mask are described. The patients were trapped following road traffic accidents and limited access prevented tracheal intubation. The laryngeal mask airway may be a useful alternative to tracheal intubation in some cases of prehospital trauma care.

Key words

Equipment; laryngeal mask.

The laryngeal mask was introduced by Brain in 1983 as a new item of equipment for the maintenance of the airway [1]. It can be inserted blind and may be used in spontaneously breathing patients or to permit intermittent positive pressure ventilation (IPPV). Although it is primarily used in routine anaesthesia [2] there have been reports of its use in emergency situations [3–5]. We believe that these are the first case reports of the use of the laryngeal mask in prehospital trauma care.

Case histories

Case 1

A 21-year-old man was the front seat passenger in a car which suffered extensive front impact damage in a head-on collision. The driver was killed instantly. The patient was trapped in an upright position by his legs, pelvis and abdomen. He had a respiratory rate of less than 10 breath.min⁻¹, a weak pulse of 110 beat.min⁻¹, and a Glasgow Coma Score of three. It was not possible to gain access to the patient in order to pass a tracheal tube. A laryngeal mask was passed from in front of the casualty, while his neck was supported in a cervical collar and assisted ventilation was successfully achieved using a bag attached to an oxygen reservoir. Following release of the patient, orotracheal intubation was attempted at the roadside but the vocal cords could not be visualised and there was considerable haemorrhage in the region of the posterior nasopharynx.

A cricothyroidotomy was performed and the airway thereby secured and protected for transfer to hospital. This

patient subsequently died from his thoracic and cerebral injuries.

Patient 2

A 32-year-old man was trapped by his lower limbs in a car. On examination, his airway was compromised by bleeding from severe facial fractures. His respiratory rate was 6 breath.min⁻¹, his chest expansion was equal, he was pale and had a pulse rate of 120 beat.min⁻¹. He was unresponsive to painful stimuli. Access to the casualty was limited by vehicle damage and attempts to control the airway and assist ventilation through an oropharyngeal airway using bag and mask technique were unsuccessful. A size 3 laryngeal mask was passed and artificial ventilation was successfully achieved. Intravenous fluids were administered and IPPV was continued during extrication of the patient, which took 30 min. Following release the patient's trachea was intubated at the roadside and he was transferred to hospital with controlled ventilation and a protected airway. After arrival in hospital he underwent emergency laparotomy and was found to have a severely lacerated liver. He died from overwhelming intra-abdominal haemorrhage which could not be controlled during surgery.

Discussion

The main objection to the use of the laryngeal mask in the emergency situation is that it does not protect the airway from aspiration of stomach contents or from haemorrhage from above. However, a recent paper has demonstrated that the mask does have a limited ability to protect the

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airway from blood which collects in the pharynx [6]. In prehospital care there are occasions when access to the patient is so limited that it is impossible to pass a tracheal tube. The alternative technique of respiratory control, using an oral airway, bag and mask, is also difficult to perform when access is limited and to maintain during the period of extrication. The laryngeal mask has the advantage that it may be passed from in front of the patient, and in these two cases the neck was maintained in a neutral position in a cervical collar. This is important in trauma resuscitation when there is a possibility of cervical spine injury and secondary damage to the spinal cord must be prevented. Although both of the patients described subsequently died from their injuries, in each case it was possible to maintain the airway until a more definitive method of control and protection was achieved to permit safe transfer to hospital. We suggest that all those involved in prehospital care should be aware of the technique of laryngeal mask insertion, which we have found to be a useful adjunct

in the management of some patients who are trapped following their accidents.

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CASE REPORT

Failed extradural anaesthesia for Caesarean section. Complication of subsequent spinal block

G. N. BECK AND A. G. GRIFFITHS

Summary

Two cases of unexpected high spinal anaesthesia following failed extradural anaesthesia for Caesarean section are described. In both cases rapid and unexpected advance of blockade, after the subarachnoid injection of moderate doses of local anaesthetic, required tracheal intubation. In one of the cases 15 ml of 0.9% saline, but no local anaesthetic, had been injected into the extradural space, suggesting that the mechanism involved is the cephalad displacement of the cerebrospinal fluid by extradural fluid, and not leakage of extradural anaesthetic solution into the subarachnoid space.

Key words

Anaesthetic techniques, regional; spinal, extradural. Complications.

Extradural anaesthesia is a popular technique for Caesarean section in the United Kingdom. Inadequate analgesia occurs in about 50% of women undergoing Caesarean section under extradural block with 0.5% bupivacaine [1–3]. Usually, supplementation with inhalational, systemic or local analgesia is adequate. However, in some cases of failed extradural blockade, further regional or general anaesthesia may be required.

In the two patients described below, spinal anaesthesia was performed following failure to establish adequate extradural anaesthesia for Caesarean section. In both excessive spinal blockade required tracheal intubation.

Case histories

Case 1

A 19-year-old primigravida (weight 52.6 kg, height 150 cm) was admitted for induction of labour with poor weight gain (2.6 kg) and postdates (term + 7 days). Artificial rupture of membranes was performed and an intravenous infusion of oxytocin started.

Three hours after induction of labour, the patient requested extradural analgesia. The extradural was sited at L_{2/3} interspace, with the patient in a sitting position, using the loss of resistance to air technique. The extradural space was identified at 4 cm, and 3 cm of catheter was introduced

into the space. Following an uneventful test dose (4 ml of 2% lignocaine), 8 ml of 0.25% bupivacaine was given. This produced a bilateral sensory block to T₈.

Two hours after siting the extradural, vaginal examination revealed a head presentation and, it was decided to proceed to Caesarean section. The patient requested to remain awake, so 10 ml of 0.5% bupivacaine was given through the extradural catheter.

After a further 10 min, the sensory block only extended to T₁₀, so a further dose of 10 ml of 0.5% bupivacaine was given. After a further 20 min the block had still not extended above T₁₀, and as the patient still wanted to stay awake, a spinal block was performed. The patient was positioned in the right lateral position, routine lumbar puncture at the L_{3/4} interspace was performed with a 26-gauge needle, and 2.0 ml of 0.5% bupivacaine with 8% glucose was injected into the cerebrospinal fluid. The patient was returned to the supine position with a wedge under the right hip. Four litres per minute of oxygen was administered by face mask. Within 5 min the block extended to T₆, and the Caesarean section was started. The patient's arterial pressure decreased to 70/40 mmHg, but was corrected with a total of 15 mg of ephedrine given intravenously.

Five minutes after starting the Caesarean section, a live baby girl was delivered (Apgar score 9 at 1 min and 10 at 5 min). At this moment the patient started to complain of

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tingling in her hands and difficulty breathing. Power in the upper limbs was normal and the SpO_2 remained over 96%. Ventilation appeared adequate. After a further 2 min the patient had increased difficulty breathing and was unable to talk. The arterial pressure was 110/70 mmHg, heart rate 120 $\text{beat}\cdot\text{min}^{-1}$ and SpO_2 97%.

In view of the respiratory difficulty, general anaesthesia was induced. Following pre-oxygenation, thiopentone 150 mg and suxamethonium 75 mg were given intravenously and cricoid pressure applied. The patient's trachea was intubated, and her lungs ventilated with 33% oxygen, 66% nitrous oxide and 0.2% to 0.4% enflurane without additional neuromuscular blockade.

At the end of the procedure (a further 50 min) the patient was allowed to breathe spontaneously 100% oxygen and woke up, whereupon her trachea was extubated.

Immediately following extubation the sensory block was to T_1 , and the patient was awake, pain free and talking. She was observed on the ward, and the block regressed over the next 3 h.

Case 2

A 33-year-old woman (weight 69.5 kg, height 150 cm) was admitted for elective Caesarean section at 39 weeks' gestation. The patient had undergone a previous Caesarean section under general anaesthesia and she requested extradural anaesthesia for this delivery.

One litre of compound sodium lactate solution was infused intravenously and, with the patient sitting, the extradural space was identified by loss of resistance to saline technique at the $L_{2/3}$ interspace. As it was not possible to thread the catheter into the extradural space, 5 ml of 0.9% saline was injected. The catheter could still not be passed. Further attempts to site the extradural at $L_{1/2}$ and $L_{3/4}$ were made. At each space the extradural space was identified, but it was not possible to pass the catheter into the space. During these attempts a total of 15 ml of 0.9% saline was injected into the extradural space to try to 'open it up'.

The patient still expressed a strong desire to remain awake for the operation, so it was decided to proceed to spinal anaesthesia. After a further circulatory preload of 1 l compound sodium lactate solution, lumbar puncture was performed in the sitting position. A 26-gauge needle was introduced via the $L_{3/4}$ interspace, and 2.5 ml of 0.5% bupivacaine with 8% glucose was injected without barbotage into the cerebrospinal fluid. After 1 min in the sitting position the patient was placed in the left lateral position. Within 5 min the block had extended bilaterally to T_3 and arterial pressure decreased to 90/60 mmHg. Ephedrine 3 mg was given intravenously and slight head up tilt was applied in an attempt to reduce further extension of the advancing block. The Caesarean section was started, and oxygen 4 l $\cdot\text{min}^{-1}$ was given by Hudson mask. Ten minutes after the insertion of the spinal, the patient started to complain of tingling in her arms and difficulty breathing; she was able to speak and cough, but was distressed by the dyspnoea. The SpO_2 remained at 99%. The arterial pressure had again decreased (to 74/40 mmHg), so ephedrine 6 mg was given intravenously.

Over the next 5 min the patient became more dyspnoeic and distressed. Following pre-oxygenation, general anaesthesia was induced with thiopentone 250 mg and suxameth-

onium 100 mg. Cricoid pressure was applied and the patient's trachea intubated. General anaesthesia was maintained with 50% oxygen, 50% nitrous oxide, 0.5% isoflurane and intermittent positive pressure ventilation. Immediately after induction a live female infant was delivered (Apgar score 7 at 1 min and 8 at 5 min). No further muscle relaxant was required for 30 min, but a total of 60 mg of intravenous ephedrine and 3500 ml intravenous fluids (2700 ml crystalloid, 800 ml colloid) was required to maintain the arterial pressure. Thirty minutes after induction of general anaesthesia diaphragmatic movement started, and atracurium 15 mg was given.

At the end of the operation, 58 min after induction of general anaesthesia, 0.5 mg glycopyrronium and 2.5 mg neostigmine was given to reverse neuromuscular blockade. Spontaneous ventilation returned and the patient was allowed to breathe 100% oxygen via the tracheal tube until she awoke, when her trachea was extubated. At this time she had good power in both hands, and sensory block was to T_2 . The block regressed over the next 2 h.

Discussion

Several drugs have been used for subarachnoid block for obstetric patients. Isobaric 0.5% bupivacaine [4] and hyperbaric 5% lignocaine [5] have been shown to be too unpredictable in obstetric patients. Currently, 0.5% bupivacaine with 8% glucose is the only drug licensed for subarachnoid block in the U.K. The ideal dose of this drug for Caesarean section remains uncertain. However, the use of less than 2 ml is associated with a substantial risk of inadequate block [6–8]. The use of 2.5 ml has been reported to be satisfactory in several series [9–12].

It has been shown that there is no relationship between the extent of block, following the subarachnoid injection of hyperbaric bupivacaine, and patient height, body mass index or length of vertebral column [13, 14]. We thus suggest the excessive blockade in these two patients, following spinal anaesthesia with normal doses of 0.5% bupivacaine (2 ml and 2.5 ml), was not as a direct result of the patient's short stature.

It has been postulated that the spinal canal acts as a fixed compartment, and that injection of fluid into the extradural space in the lumbar region results in cephalad displacement of the cerebrospinal fluid and relative collapse of the subarachnoid space below the termination of the spinal cord [15, 16]. This collapse may result in failure of spinal blockade if the spinal needle fails to enter the subarachnoid space [16]. If, however, dural puncture is successful, a given volume of injected local anaesthetic may then spread more extensively as a result of the reduced cerebrospinal fluid volume in the lumbar region.

Over recent years there have been several reports of combined spinal and extradural techniques for Caesarean section [17–21]. These techniques have not been associated with unexpected excessive high spinal blockade. However, a planned combined technique differs from the two cases we describe in two important ways. Firstly, in a combined technique, the subarachnoid injection is made prior to any extradural injection. Thus the local anaesthetic may, at least in part, be 'fixed' [22] prior to displacement of the cerebrospinal fluid by the extradural injection. Secondly the volume of both extradural and subarachnoid drugs tends to be lower in a planned combined block.

An extensive literature search has revealed only one similar case [23]. In this case 33 ml of 0.5% bupivacaine had been injected down an extradural catheter over 80 min prior to a spinal block with 1.6 ml of 0.5% bupivacaine with 8% glucose. Tracheal intubation was required as a result of an excessive block.

It has previously been suggested that diffusion of local anaesthetic solution across the dural puncture site into the cerebrospinal fluid may cause unexpected high blockade after accidental dural puncture [24–29]. This mechanism could explain the one previous report [23], and our first case. However, in our second case only 0.9% saline, and no local anaesthetic, had been injected into the extradural space. We would therefore suggest that the excessive blockade is a physical effect of the volume of fluid in the extradural space and does not require the presence of extradural local anaesthetic solution.

In a recent review article on spinal anaesthesia [30], Kestin recommended the use of spinal anaesthesia for Caesarean section if extradural blockade is inadequate following the maximum dose of local anaesthetic. However, in view of the cases we report, and the previous report of excessive blockade following this technique, we would advise that extreme caution is required in performing spinal anaesthesia in the presence of any fluid in the extradural space. This is, of course, particularly important in a patient where difficulty in tracheal intubation is anticipated.

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An automated system for testing the accuracy of patient-controlled analgesia devices

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Summary

A system was developed to test the accuracy of patient-controlled analgesia devices in situations simulating clinical use. Bolus requests are made automatically at predetermined intervals, and the infusate delivered is measured and recorded without the need for operator presence. To ensure clinical relevance, the bolus request times used in this study corresponded to a pattern typical of those requested by patients on the ward. Graseby, Abbott Provider 5500 and IVAC patient-controlled analgesia devices were tested and found to deliver reasonably accurately over a 24 h period. However, when an infusion was started in an unprimed system or after a period of no bolus requests in a bolus-only mode the Graseby and IVAC machines under-delivered. This system provides a means of testing patient-controlled analgesia devices operating in any delivery mode.

Key words

*Analgesia; patient-controlled.
Equipment; infusion pumps, computer.*

Over the last 20 years, patient-controlled analgesia (PCA) has developed from an investigative tool [1] to a well-used and desirable form of postoperative pain relief [2]. The choice of suitable PCA devices has increased accordingly although ... 'many are available for use without a single publication to document the safety and utility of the device in the routine patient care situation' [3]. Where articles have been published reviewing these devices, most report their accuracy [3–6] assessed volumetrically, or more usually gravimetrically, from collection of infusate delivered. In these studies the output was measured manually, and continuous infusion and bolus doses were tested separately with no effort being made to simulate clinical use. Testing a large number of PCA devices under simulated clinical conditions would be very time consuming and to request bolus doses at timed intervals, with and without a background infusion, would be even more laborious. One solution is a computer-controlled system where bolus requests can be made by the computer and the weight of infusate delivered recorded automatically. The aim of this study was to develop a system for the automated assessment of PCA devices with particular attention to their accuracy in a simulation of clinical use. Three types of electronic PCA devices were studied using this system.

Methods

A T1000 lap-top computer (Toshiba corporation, Tokyo, Japan) was used to control a system which could initiate demands and gravimetrically measure the fluid delivered by the PCA devices. Narrow bore plastic tubing connected the PCA device to a long 14 G needle which rested on the bottom of a measuring cylinder below a layer of light oil introduced to reduce inaccuracies due to evaporation. Fluid delivered by each PCA device was continuously weighed on a calibrated Mettler PM2000 electronic balance (Mettler Instruments, AG Zurich, Switzerland). The computer was programmed to interrogate the balance hourly; the weight and time were then automatically recorded on computer disk. The computer program simulated the bolus requests of a patient via a purpose-built interface box connected to the PCA device. When a bolus was requested the interface box activated the PCA device either directly by an electrical connection or by a pneumatic connection (for the Graseby PCAS). The pneumatic pressure was provided by a Maxima pneumatic aquarium pump (Ralph C. Hagen, Leeds, UK). Communication was via serial connections made between the computer and interface box and between the computer and the electronic

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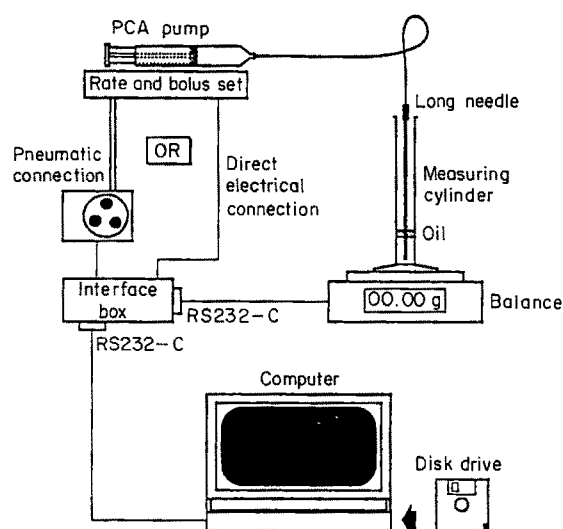


Fig. 1. A diagrammatic representation of the test system.

balance (RS232-C) routed through the interface box. The system is illustrated in Figure 1.

Using this system an assessment was made of the accuracy of 12 Graseby PCAS devices (Graseby Medical, Watford, UK), some of which had been in service in our hospital for the past 5 years. Three new Provider 5500 pumps (Abbott Ambulatory Infusion Systems, San Diego, USA) and one new IVAC PCAinfuser (IVAC Corporation, San Diego, USA) were also tested.

Each PCA device was set up to deliver water in three different delivery modes: (1) bolus only: 1 ml bolus doses according to requests from the computer; (2) infusion only: 1 ml.h⁻¹ infusion, no bolus requests; (3) bolus + infusion: 1 ml.h⁻¹ infusion plus 1 ml bolus doses according to requests from the computer.

Each PCA device was studied for 24 h in each delivery mode.

To produce results more representative of the use of PCA devices in the wards, the timing of bolus requests was based on clinical observations of patients using PCA for postoperative pain relief. The chosen profile is illustrated in Figure 2.

The weight of infusate (water) delivered was converted to a volume by dividing by the density of the water at the temperature in the laboratory (0.997 g.ml⁻¹). Data were analysed using the Supercalc 4 spreadsheet programme where the accuracy of hourly delivery in the three modes was expressed as a percentage of the expected volume delivered. Statistical analysis was carried out on the data from the Graseby PCAS devices. Differences in accuracy among the three modes of delivery were compared using repeated measures analysis of variance. The data from the other devices were insufficient to warrant statistical analysis.

To determine whether results may have been affected by evaporation of infusate, balance drift, or other equipment artifacts, the system was operated for 10 24 h periods during which no fluid was delivered.

Results

When the system was run with no infusate being delivered the mean fluctuation was 0.00g (SD = 0.08, range -0.09 g to +0.14 g) over a 24 h period. In the worst possible case of

Table 1. Accuracy of PCA pumps in all three delivery modes.

		Bolus (% of nominal volume delivered)	Infusion (% of nominal volume delivered)	Bolus + infusion (% of nominal volume delivered)
Graseby	0-1 h	88.3	51.9	88.7
(average)	1-24 h	98.9 (1.72)	97.6 (1.75)	98.3 (0.88)
Provider	0-1 h	107.5	102.6	99.3
(average)	1-24 h	101.5 (1.18)	99.4 (1.52)	99.3 (1.38)
IVAC	0-1 h	98.8	81.2	96.1
	1-24 h	95.9 (9.83)*	97.3 (1.71)	97.8 (6.87)*

The values for the 12 Graseby PCAS and three Provider 5500 pumps are averaged. The values shown for the 1-24 h periods are expressed as mean (standard deviation). *These large standard deviations are explained in the discussion.

the maximum fluctuation occurring in the infusion-only mode (smallest amount delivered) this would only lead to a discrepancy of 0.58%. These fluctuations were therefore considered insignificant.

With all the devices tested there was little difference in the hourly accuracy except in the first hour (Table 1). The Graseby PCAS and IVAC PCAinfuser both underdelivered and the Abbott Provider 5500 overdelivered during this time. There was also a transient period of underdelivery with the Graseby and IVAC pumps following the 7 h period of no bolus requests, but delivery did not fall below 90% of that which was expected (see Fig. 3). The Abbott Provider 5500 delivered relatively accurately over the remaining 23 h period. If the two periods of inaccurate delivery described are excluded, then the average delivery was generally within 3% of expected in all modes. The exceptions were two of the Graseby PCAS devices which gave mean delivery rates of 95.7% and 94.2% in the infusion-only mode.

Statistical analysis of the results for the Graseby PCAS device showed a difference among the modes in both the first hour and in the 1-24 h periods, the infusion-only mode giving the greatest underdelivery in both cases.

Discussion

A fully automated system has been developed which can be used to test PCA devices. The system has been used to test

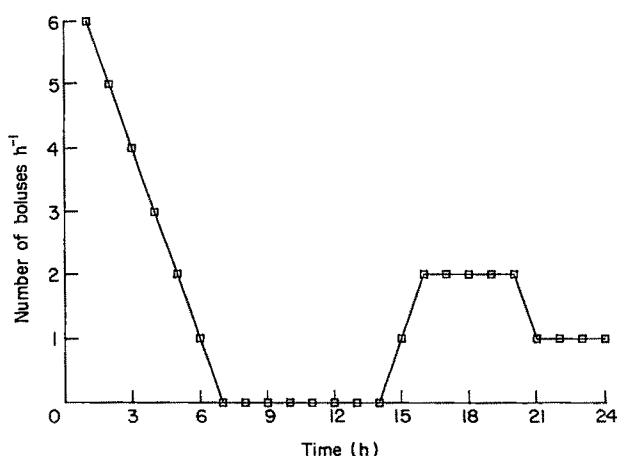


Fig. 2. Demand rate profile used to test PCA devices. The patient has a high request rate initially which declines to zero at night. An early morning peak is usually seen which settles to a low request rate.

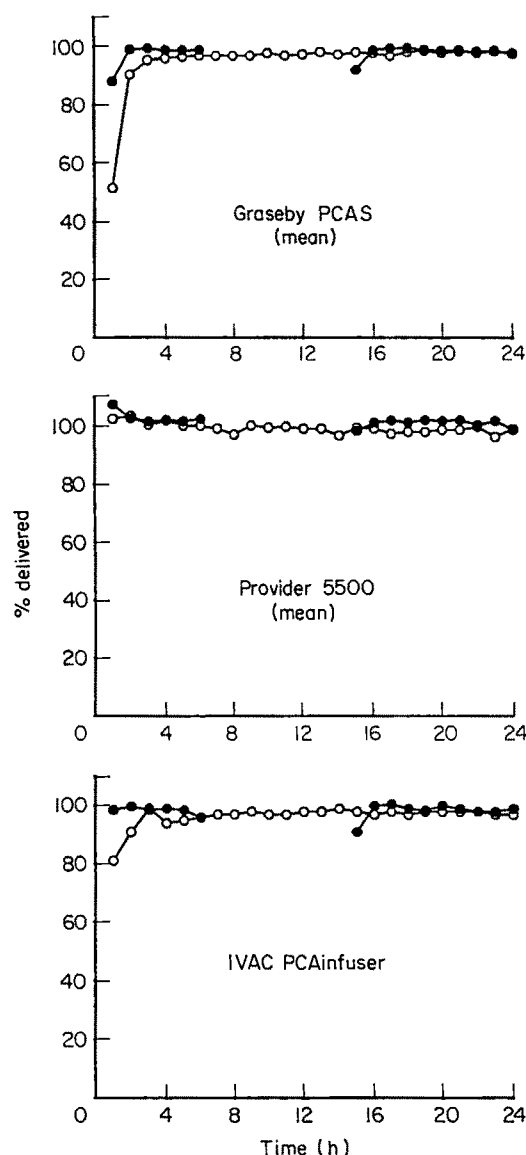


Fig. 3. The graphs show the actual volume delivered/expected volume delivered as a percentage for each hour. Only bolus (●) and infusion (○) modes are shown for clarity.

a number of devices which were shown to be reasonably accurate i.e. within 10% of the bolus or continuous infusion dose selected [7]. In particular they were shown to be accurate in a clinically relevant delivery mode. However, unprimed syringe driver systems could underdeliver by more than 10% in the first hour.

It has been suggested that bolus volumes should be accurate to within 10% of the selected dose. With continuous infusion modes the volumes should be accurate to within 5% between flow settings of 5–50 ml.h⁻¹ and 10% for all other settings [7]. It is surely more important that accuracy is greatest at flow rates used clinically (e.g. 1 ml.h⁻¹ in our centre) especially as more concentrated solutions are used and thus discrepancies can have greater clinical effects.

The low volumes delivered in the first hour by the Graseby pumps, and to a lesser extent the IVAC pump, can be explained if we consider the method used to set up the PCA devices. A study was made of the method used by the nurses in our hospital in an effort to reflect further clinical

use of the devices. It was found that they first primed the syringes by hand prior to placing them in the pump and we decided to use this same method in the knowledge that priming the system using the pump itself may have given a more accurate delivery in the first hour. However, if the syringe later required changing on the ward the latter approach would not be possible without either disconnecting the infusion or giving the patient a bolus dose.

The reason for underdelivery in the hour following the 7 h period of no bolus requests is unclear. One explanation could be the development of increased compliance in the delivery system which has to be overcome prior to accurate delivery rates being achieved.

The significantly different accuracy of the Graseby PCAS in the three modes is likely to be due in part to the differing nominal values of infusate delivered. In the first hour only 1 ml was delivered in the infusion-only mode compared to 6 ml and 7 ml in the bolus-only and combined modes respectively. This would lead to less inaccuracy in the latter two delivery modes, as was observed (Table 1), as there would be greater tendency for the compliance changes and priming differences to be overcome within that hour. In the 1–24 h period, although the difference in the delivery between the modes was statistically significant, the actual percentage differences were small and clinically unimportant.

The accuracy of the Abbott Provider 5500 is probably due to the fact that it operates as a peristaltic pump rather than a syringe driver. The system must be primed using the pump mechanism and there seems to be no effect of increasing compliance.

On testing the IVAC PCAinfuser it was found that in the second hour there was a failure to give one of the requested bolus doses, hence the large standard deviation value in the 1–24 h period. The software in this device limits bolus requests to a maximum of 10 in any 2 h period. The reason behind this may be that if any more than 10 requests are made then the manufacturers believe that there should be a review of the prescription. The number of bolus doses requested and the number given over the previous 24 h are displayed thereby giving an indication of a prescription problem. However, in the opinion of the authors, this arbitrary limit should not be included in the software. When a lockout period is chosen both prescriber and patient should be able to expect a bolus dose to be available if that period is exceeded. If a large number of requests are made then dosages should be reviewed but not limited by the PCA device. The visual display of requests on the device is a useful option.

The system described has been shown to be useful in the testing of the accuracy of PCA devices and it has revealed some idiosyncrasies of PCA pump design. It will be possible to test all PCA devices available through appropriate modifications to the interface where required. This system is reliable, fully automated and could well be used in the future to test all PCA devices in a clinically relevant manner.

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Laryngeal mask and anaesthetic waste gas exposure

P. LAMBERT-JENSEN, N. E. CHRISTENSEN AND J. BRYNNUM

Summary

In recent years there has been a growing awareness of the possible hazards caused by anaesthetic gases in operating theatres. The laryngeal mask airway provides an alternative both to tracheal intubation and the face mask although the implications for operating theatre contamination have not been quantified. This paper describes the incidence and magnitude of exposure of theatre personnel to waste anaesthetic gases during laryngeal mask airway anaesthesia. The leakage of anaesthetic gases to the anaesthetist's breathing zone was monitored using a Brüel & Kjaer Multi Gas Monitor, Type 1302 during 50 general anaesthetics employing either spontaneous ($n = 24$) or controlled ($n = 26$) ventilation. All patients were anaesthetised with propofol, alfentanil and nitrous oxide. There was no statistically significant association between the amount of anaesthetic gas leakage and ventilation method. The laryngeal mask airway meets occupational safety requirements on nitrous oxide concentrations in the operating theatre environment.

Key words

Equipment; laryngeal mask airway.

Anaesthetic gases; pollution, occupational exposure.

There is growing concern about the pollution of the operating theatre environment with anaesthetic gases and vapours. In 1977 the National Institute For Occupational Safety and Health (NIOSH) proposed a time-weighted average (TWA) occupational exposure limit of 25 parts per million (ppm) for nitrous oxide [1]. However, maximal exposure limits for nitrous oxide have been suggested or agreed upon only in some countries, notably the Netherlands (25 ppm), the USA, (50 ppm), and Sweden, Norway, Denmark, and Italy (100 ppm) [2–4].

The laryngeal mask airway is a new kind of airway [5]. Commercially available since 1988, it is now being widely used as an alternative to tracheal intubation and the face mask. The laryngeal mask is reportedly gas-tight in positive pressure ventilation up to 3 kPa [6] although clinical evidence is not available. The mask tightness in experimental laryngeal mask anaesthesia of seven patients during spontaneous ventilation has previously been shown to match that of tracheal anaesthesia [7].

The present study reports the incidence and magnitude of anaesthetic gas exposure during laryngeal mask anaesthesia during either spontaneous or controlled ventilation. The aims of the study were to examine the relationship between N_2O leakage and the ventilation technique, measure nitrous oxide concentrations in the anaesthetist's

breathing zone, and to examine possible correlations between N_2O leakage, sex and weight.

Method

Approval of the study was obtained from the local ethics committee and all patients gave their informed consent. Fifty three patients conforming to ASA 1, scheduled for elective arthroscopy of the knee were randomly allocated to one of two groups — spontaneous respiration (group 1) or controlled ventilation (group 2). The patients were unpremedicated and intravenous access was established after arrival in the anaesthetic room. The ECG, end-tidal carbon dioxide concentration, respiratory rate and peripheral oxygen saturation were monitored continuously during the peri-operative period; every third minute the arterial blood pressure and heart rate were recorded using a Hewlett Packard Component Monitoring System.

After making baseline readings, anaesthesia was induced with propofol $1.5\text{--}3\text{ mg.kg}^{-1}$ (until loss of eyelash reflex) and maintained with propofol, alfentanil and nitrous oxide 66% in oxygen. The laryngeal mask was inserted by one of three experienced anaesthetists using the technique recommended by the manufacturers. A size 4 laryngeal mask was

used for either sex. Thirty-five ml of air were injected into the cuff following the manufacturer's recommendations.

The patient's lungs were ventilated via a Hafnia A system using a fresh gas flow of 100 ml.kg^{-1} [8]. Controlled ventilation was performed by manual compression of the reservoir bag. The mean inspiratory pressure was measured continuously and the peak inspiratory pressure was determined at 5 min intervals, using a water-filled catheter within the laryngeal/mask, passed through a tube connector incorporating a rubber seal. The tip of the catheter was placed at the level of the aperture bars and the other end connected to a Medex MX860 pressure transducer.

Nitrous oxide concentrations within the anaesthetist's breathing zone were measured with a Brüel & Kjaer Multi-gas Monitor Type 1302 by a technique known as photo-acoustic spectroscopy. The sampling hose was attached 0.3 m behind and 0.3 m above the patient's head, a point considered to be within the anaesthetist's breathing zone. Measurements began 1 min after positioning the laryngeal mask and samples were taken at 5 min intervals until the end of surgery.

A prestudy test of the anaesthetic equipment fully satisfied official safety regulations and all the apparatus was meticulously checked according to our standard protocol.

The anaesthetic room had a volume of 92.93 m^3 and the room ventilation was $1032 \text{ m}^3.\text{h}^{-1}$, approximately 11 air changes each hour. The active scavenging system (ejector flowmeter) at the waste gas outlet of the anaesthetic machine had a capacity of 15 l.min^{-1} .

Data were analysed using unpaired Student's *t*-tests and Wilcoxon's tests, linear regression and Goodman-Kruskal's gamma tests. Statistical significance was assumed at $p < 0.05$.

Results

A total of 53 patients were admitted to the study. Three patients were subsequently excluded because a size 3 laryngeal mask was used ($n = 2$) and because of leaks in the Hafnia A system which were discovered postoperatively ($n = 1$). There were no significant differences between the two groups in terms of demographic data. All three anaesthetists were experienced physicians, trained in inducing and maintaining laryngeal mask anaesthesia. Accordingly, the mask was successfully introduced on the first or second attempt in all patients. In two patients there was an audible leak and the mask was repositioned. There were no significant differences between the results obtained by the three anaesthetists.

Ventilation of the 50 patients included in the study was acceptable as judged from end-tidal carbon dioxide concentrations and peripheral oxygen saturations and none of the spontaneously breathing patients needed any supplemen-

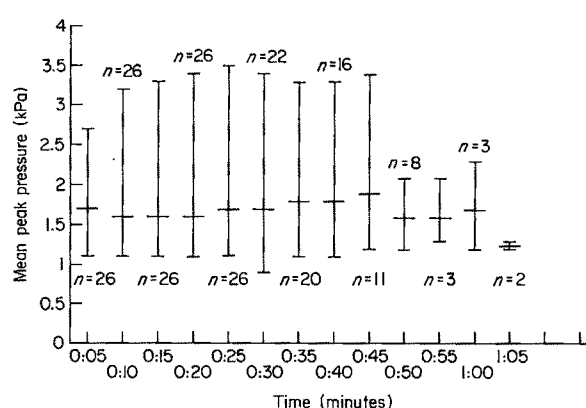


Fig. 1. Mean peak inspiratory pressure with ranges.

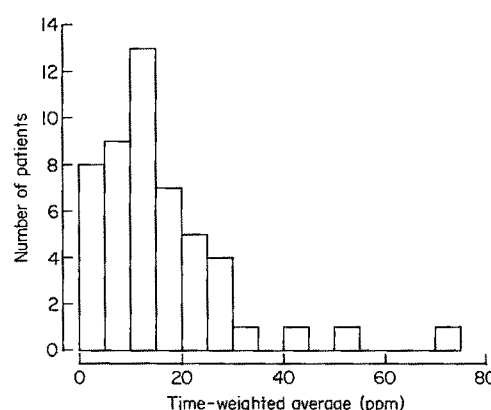


Fig. 2. N_2O leakage as time-weighted average.

tary ventilation. The inspiratory peak pressure was below 2.3 kPa in 25 of the 26 patients in controlled ventilation (Fig. 1).

On three occasions, the nitrous oxide concentration exceeded 40 ppm (TWA); one was associated with an inspiratory peak pressure of 3.3 kPa and another was due to a minor cuff leak. There is no ready explanation for the third case.

The time weighted average values were unevenly distributed (Fig. 2) with many small and a few large values. Patients having controlled ventilation had a larger N_2O leakage than patients breathing spontaneously (Table 1), but this difference was not statistically significant. The 25 ppm limit was exceeded in eight cases; six of these occurred in patients having controlled ventilation and although there was no significant difference in the TWA distribution between the sexes, seven of the eight were men. The presence of large N_2O leaks was unsuspected — there were no audible indications and they were only detected by the gas monitoring device.

The average leak of N_2O recorded during the first 5 min periods (Table 2) did not vary significantly between the two

Table 1. Measured N_2O under spontaneous and controlled ventilation; time-weighted average (parts per million); 25, 50 and 75 percentile.

	Spontaneous	Controlled	Total
Females	4.1, 13.6, 16.6	8.6, 12.7, 16.2	6.1, 13.4, 16.1
Males	8.3, 16.0, 18.9	6.6, 12.1, 30.1	7.3, 15.4, 26.0
Total	7.5, 14.2, 18.4	7.0, 12.4, 24.0	7.0, 13.8, 20.6

Table 2. Measured N₂O under spontaneous and controlled ventilation; average during the first five 5 min periods (parts per million). 25, 50 and 75 percentile.

	Spontaneous	Controlled	Total
Females	8.0, 11.0, 14.4	7.4, 13.1, 17.5	8.1, 12.8, 15.9
Males	8.2, 15.2, 28.2	6.6, 13.8, 38.2	7.3, 14.7, 30.1
Total	8.1, 13.2, 18.6	7.0, 13.7, 24.5	8.2, 13.6, 22.1

Table 3. Nitrous oxide exceeding 25 ppm during the first five 5 min periods.

Ventilation	Numbers of values over 25 ppm						Total
	0	1	2	3	4	5	
Spontaneous	16	4	3	1	0	0	24
Controlled	14	4	1	4	1	1	26
Total	30	8	4	5	1	2	50

types of ventilation and again there was no statistical difference between the sexes.

The number of N₂O values above the 25 ppm limit within the first five 5 min periods (Table 3) did not vary

significantly between the two ventilation groups and again gender had no influence.

The relationship between time and mask seal is described in terms of the size of gas leak as a function of time, with log N₂O against time and average values plotted as a curve (Fig. 3). Curves were approximated by straight lines and the distribution of the slopes was analysed.

Discussion

Our reference for gas leakage was the NIOSH recommendation of a maximal time-weighted average occupational exposure to nitrous oxide of 25 ppm. This limit is lower than the Danish national limit of 100 ppm measured as a

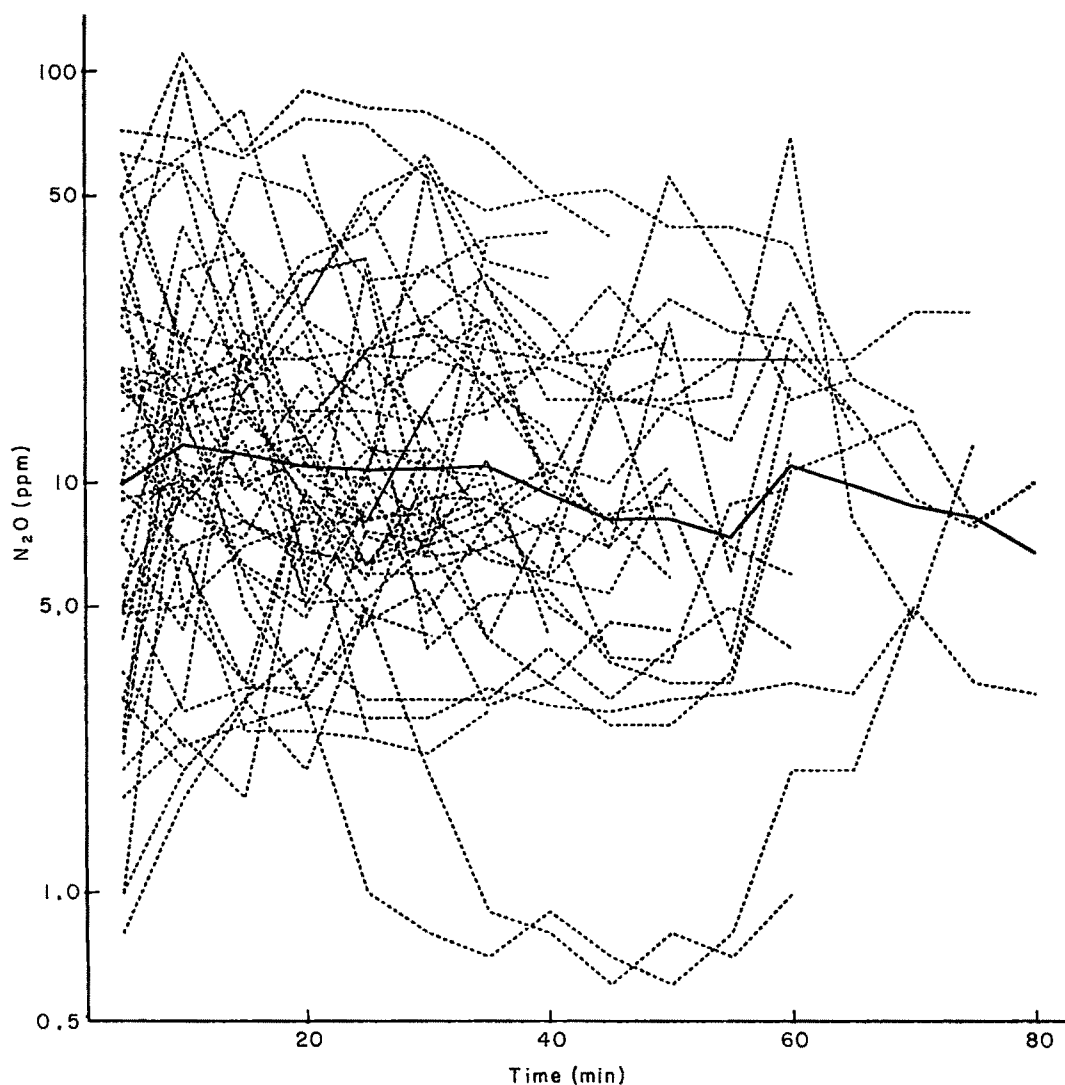


Fig. 3. The size of N₂O gas leakage for each of the 50 patients as a function of time and log N₂O concentration.

TWA of an 8-h workday where any single exposure must never exceed a level of 200 ppm, not even within very short working periods.

In a previous experiment on seven patients the integrity of laryngeal mask sealing during spontaneous ventilation was sufficient to limit the N_2O values to less than the 25 ppm limit proposed by the NIOSH; one case, however, had considerable leakage of N_2O during positive pressure ventilation. We have demonstrated that positive pressure ventilation is possible up to 2.3 kPa, which is in agreement with studies by Maltby and colleagues [11]. According to Brain [9] an inspiratory peak pressure of up to 3 kPa may be safely applied. An investigation of 100 patients who had a laryngeal mask size 3 showed a mean leak pressure of 1.7 kPa [10].

The low pressure seal formed above the laryngeal inlet by the inflated cuff is affected by factors such as patient size, anatomical variations, depth of anaesthesia and the inspiratory pressure used. We have found that the anatomical structures of the hypopharynx apparently adjust to the laryngeal mask so that any audible leakage disappears within 1–5 min.

We had expected that the mask seal would improve during anaesthesia, but slope estimates plotted as a mean slope in a coordinate system (Fig. 3) showed that the mask tightness was virtually unchanged. A possible explanation for this is that the integrity of the seal becomes disrupted during the 'lightening' phase towards the end of the operation.

This paper shows that the use of laryngeal mask airways (size 4) in spontaneously breathing or ventilated patients is not associated with high concentrations of nitrous oxide in the anaesthetist's breathing zone.

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The upgraded Finapres 2300e

A clinical evaluation of a continuous noninvasive blood pressure monitor

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Summary

Measurements from the upgraded Finapres 2300e continuous noninvasive blood pressure monitor, the Finapres 2300 and Colin oscillometric noninvasive blood pressure monitor were compared with invasive arterial line blood pressure readings. Fifteen young Chinese patients undergoing elective spinal surgery of more than 2 h duration had contemporaneous blood pressure measurements digitally recorded every minute. Data were analysed using methods described by Bland and Altman to assess agreement of noninvasive devices with an arterial line. Results showed that although the Finapres 2300e was significantly more accurate than the Finapres 2300 and Colin noninvasive blood pressure monitors it could not be recommended as a substitute for continuous arterial line blood pressure monitoring. Both Finapres devices demonstrated reductions in accuracy related to time (drift) and over-read diastolic and mean pressures by 5–8 mmHg throughout the range of mean arterial line pressures (bias). The Colin was consistently less accurate than the Finapres monitors and performed worst at low mean arterial line pressures.

Key words

*Blood pressure; measurement.
Monitoring; blood pressure, noninvasive.
Equipment; blood pressure, Finapres.*

Continuous monitoring of arterial blood pressure is often desirable [1, 2] and is widely practised during anaesthesia. Arterial cannulation has associated morbidity [3, 4] and noninvasive alternative may be more appropriate. This alternative device should meet equivalent standards of reliability and accuracy before recommendation as a substitute for invasive blood pressure monitoring [5].

The Finapres (model 2300; Ohmeda, Denver) noninvasive continuous blood pressure monitor became commercially available in 1989. Initial studies in the clinical environment were favourable [6] but recently performance has been criticised in three specific areas: accuracy in long-term use (drift), poor agreement between the Finapres and arterial line mean/diastolic pressures [7, 8], and unpredictable variability between the arterial line and the Finapres [9, 10].

A second generation Finapres (model 2300e) has been designed to improve accuracy. The aim of this study was to compare the performance of the upgraded Finapres device with the original version, particularly as regards oscillometric and direct arterial blood pressure measurements.

Methods

Fifteen young Chinese patients scheduled for elective corrective spinal surgery were admitted to the trial. The

study was approved by the Faculty of Medicine Ethics Committee (University of Hong Kong) and informed consent was obtained from all patients or their parents. Only patients weighing more than 30 kg and undergoing arterial cannulation as a routine component of their anaesthetic technique were included in the study.

Blood pressure was measured noninvasively before surgery five times in each arm (Colin Compact Multi-monitor BP-408 Mark II (Colin)) to ascertain any between-arm difference. A 22 G cannula (Insite, Deseret) was put into a radial artery and connected to a re-usable transducer (Spectramed Statham Physiological Pressure Transducer) via a 1.6 m long, 1.5 mm diameter tube. This system had an acceptable dynamic response according to the guidelines defined by Gardner [11]. The arterial transducer was adjusted to the level of the two Finapres electropneumatic transducers, connected to a Datex Cardiopac CC 104 monitor and calibrated against a 100 mmHg column and atmospheric pressure. Two small-sized Finapres cuffs connected to a 2300 (old Finapres) and a 2300e Finapres blood pressure monitor (new Finapres) respectively, were positioned over the middle phalanges of the index and middle fingers of the same arm used for measuring direct arterial pressure. A medium sized noninvasive blood pressure measurement cuff (Colin) was positioned on the arm contralateral to the arterial cannula. Any

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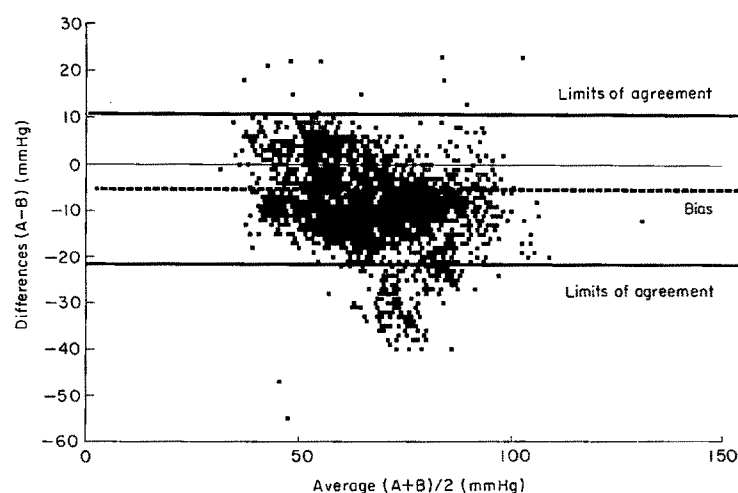


Fig. 1. Scatterplot of the upgraded Finapres and arterial line (differences against average) for systolic, diastolic and mean pressure readings showing limits of agreement and bias ($n = 15$).

difference in height between the Colin blood pressure cuff and contralateral radial artery was recorded. Data collection commenced following induction of anaesthesia and placement of the patient in the prone or lateral position for surgery.

The four pressure monitoring devices were interfaced with a Sigma PC/XT computer via an 8-port 232c multi-serial card (Decision Computers International, Taiwan). The Finapres and arterial line blood pressure and time data were stored to disc in ASCII format at 1 min intervals in real time using data acquisition software written for this system [12]. The noninvasive blood pressure monitor transmitted data at 5 min intervals and recording was continued for a minimum 2 h period. The files were imported into Lotus 123W for display and data were analysed using Minitab statistical software [13]. Statistical significance ($p < 0.05$) of the error and bias between monitors, time periods and pressure ranges was determined using Student's *t*-tests and ANOVA where appropriate.

Data analysis

Assessment of agreement between the noninvasive devices and the arterial line employed a method described by Bland and Altman [14]. The method analyses the difference between contemporaneous arterial line measurements (A) and the blood pressure readings from the device being assessed (B).

A plot of the differences between the arterial line and the noninvasive device (A-B) against their average readings $((A+B)/2)$ allows us to see the strength of agreement (Fig. 1), which can be determined by relating the standard deviation (SD) of the differences (error) to the average of all the differences (bias). The 'limits of agreement' represent the range within which 95% of the differences fall, and is the bias (2 SD) if the differences are normally distributed. To determine this data distribution, a graph of the size of difference was plotted against frequency of occurrence (Fig. 2). The monitoring devices can be considered interchangeable if the 'limits of agreement' are within (SD) 10 mmHg [9].

Linear regression of A-B against $(A+B)/2$ data indicates any relationship between the size of the difference and the

range of measurement (e.g. larger differences in the lower blood pressure range of measurement). The bias and standard deviation of the differences were also calculated for specific time periods during anaesthesia to determine the measure of agreement in relation to the duration of monitoring (i.e. drift).

Each method of data analysis was applied to the new and old Finapres models and Colin for systolic, diastolic and mean pressures both to determine group differences and for each individual patient.

Results

A total of 3000 sets of contemporaneous measurements were collected from 10 female and five male patients for analysis. The mean age of all patients was 19.8 years (range 12 to 39 years) and average body weight was 47.8 kg (range 37 to 63 kg). Seven patients were placed in the lateral position and the remainder were positioned prone. Mean duration of monitoring was 200 min (range 135 to 245 min). Pre-operative measurement of blood pressure revealed no significant difference between either arm. The data were edited to remove those readings associated with cuff repositioning and arterial line flushing. Overall bias and standard deviations of the differences from the arterial line were calculated for the noninvasive devices as described in the data analysis section.

The upgraded Finapres agreement with the arterial line was significantly better than the old Finapres and Colin ($p < 0.05$) (Fig. 3) (Table 1). Both Finapres devices had similar bias, indicating a tendency to over-read both the diastolic and mean pressures ($p < 0.05$) (Table 1). Under-reading by the Colin was demonstrated by a large positive bias for diastolic readings (Table 1).

The increasing standard deviation of differences calculated in each 30 min time period from the commencement of monitoring illustrates a decrease in strength of agreement between both Finapres devices and the arterial line (Fig. 4). Accuracy of blood pressure measurement at the commencement of data acquisition deteriorated significantly after 90 min monitoring ($p < 0.05$). The accuracy of the Finapres devices was not influenced by the magnitude of the true (arterial line) reading. The lack of relationship

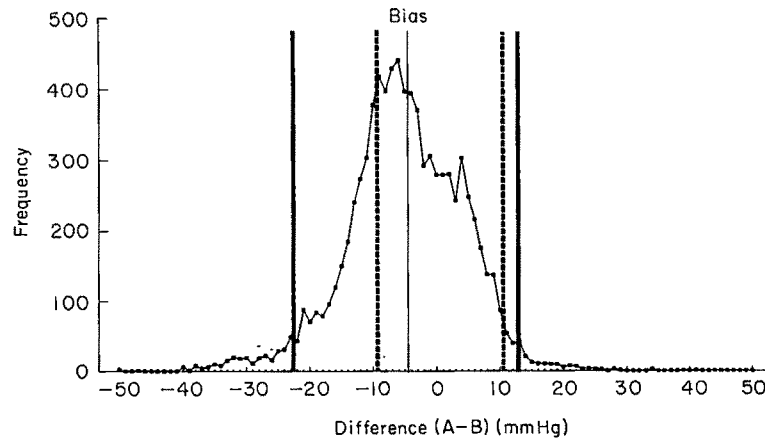


Fig. 2. Frequency distribution showing the differences between the upgraded Finapres and the arterial line for systolic, diastolic and mean pressures, demonstrating the limits of agreement (—) of the upgraded Finapres and the recommended limits of agreement (---) ($n = 15$).

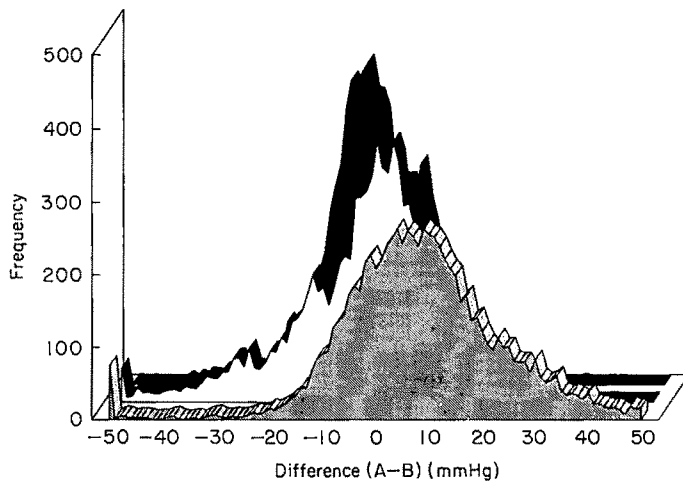


Fig. 3. Frequency distribution showing the differences between noninvasive devices and the arterial line for systolic, diastolic and mean pressures in all patients. The spread of the differences is an indicator of agreement with arterial line measurements. ▨, Colin; □, old Finapres; ■, new Finapres.

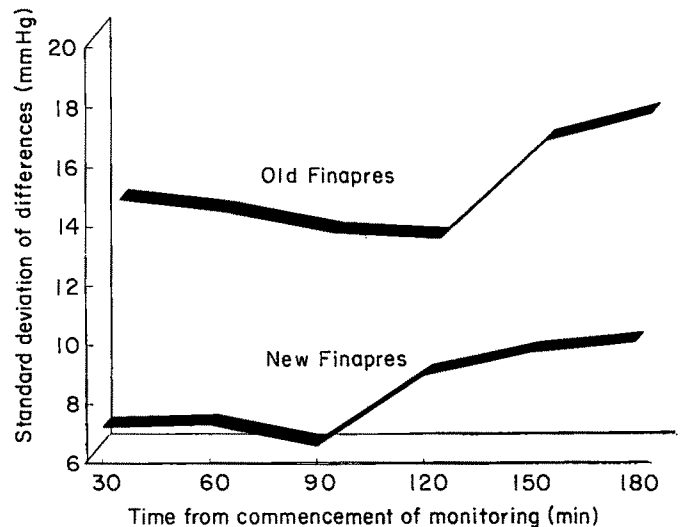


Fig. 4. Graph of the standard deviation of differences between the two Finapres monitors and the arterial line mean arterial pressure (MAP) readings against time ($n = 15$).

Table 1. Mean difference (bias) and standard deviation of the differences between the noninvasive blood pressure monitors and the arterial line systolic, diastolic and mean pressures (mmHg).

	New Finapres			Old Finapres			Colin		
	Systolic	Diastolic	Mean arterial pressure	Systolic	Diastolic	Mean arterial pressure	Systolic	Diastolic	Mean arterial pressure
Mean difference; bias (SE)	-0.57 (0.15)	-8.77 (0.16)	-5.49 (0.15)	-1.81 (0.25)	-7.81 (0.24)	-4.9 (0.24)	7.65 (0.33)	13.1 (0.28)	4.74 (0.31)
Standard deviation of differences	8.35	8.75	8.34	13.56	12.89	13.19	17.82	14.98	16.67
Mean difference; bias, systolic, diastolic and mean pressures (SE)		-4.95 (0.099)			-4.77 (0.014)			8.5 (0.018)	
Standard deviation of differences; systolic, diastolic and mean pressures		9.13			13.42			16.89	

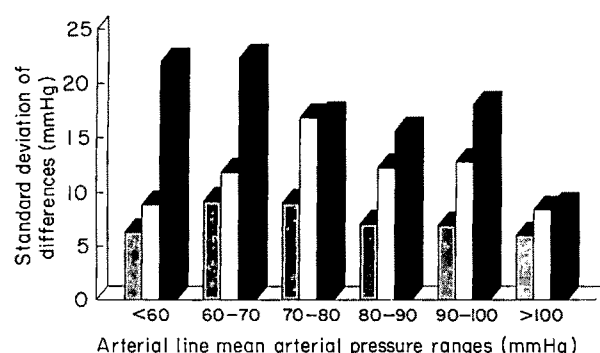


Fig. 5. Histogram of the standard deviation of the differences between the arterial line and the new and old Finapres and Colin MAP readings in various arterial line MAP ranges ($n = 15$). □, old Finapres; ▨, Colin; ■, new Finapres.

between measurement error and true value was confirmed by calculating the standard deviation of differences for systolic, diastolic and mean pressures in various mean arterial line pressure ranges (Fig. 5). The agreement of the Colin with the arterial line was poorest at low mean arterial pressures.

Examination of the standard deviation of the differences from the arterial line, after the bias was corrected to zero for each noninvasive device and for each patient individually, showed the new Finapres met the clinical standards, allowing substitution of this device for the arterial line in four patients for systolic, diastolic and mean pressures. This was possible for only one patient using the old Finapres. The mean arterial pressure readings were clinically acceptable in eight patients using the new Finapres and in four patients with the old Finapres (Table 2). The strength of agreement of the Colin with the arterial line did not meet these standards in any patient.

Discussion

This study showed that the upgraded Finapres 2300e was statistically and clinically more accurate than the old Finapres 2300 and the Colin oscillometric noninvasive blood pressure monitor, but we were unable to recommend the new Finapres as a substitute for arterial line blood

pressure monitoring, since its limits of agreement, calculated from subject pooled data, were outside the limits of clinical acceptability. Previous studies demonstrate the Finapres to be as accurate as currently employed oscillometric techniques [10, 15] but this study shows that the new Finapres 2300e is now a superior alternative. The Colin, however, was not designed to be a continuous monitor of blood pressure and it is more appropriate to compare continuous noninvasive devices with an arterial line.

Clinical data acquisition presents the Finapres with various 'adverse' monitoring conditions which may reduce its accuracy. Putting the Finapres cuff on correctly is mandatory for accurate monitoring [16], but the cumbersome nature of the Finapres pneumatic transducer and its large connecting cable makes application of Finapres cuffs (particularly the small size) to patients in the lateral and prone positions awkward and provides a potential source of operator error. To reduce this variability all cuff applications were performed by the same two anaesthetists after viewing the training video. Further investigation is currently being undertaken to determine which aspects of finger cuff application contribute towards inaccurate continuous noninvasive blood measurement. The position of the Finapres cuff relative to the right atrium has been considered in previous studies [17, 18]. Hydrostatic effect was not corrected for and our data were consistent with previous studies showing over-reading of both Finapres devices for diastolic and mean pressures by 5–8 mmHg.

The new and the old Finapres devices demonstrated a reduction in accuracy related to time. This occurred after approximately 90 min of monitoring. The new Finapres was least affected and neither it nor the old model deteriorated to the level of inaccuracy exhibited by the Colin; however, the Colin demonstrated no temporal deterioration in accuracy. The most probable cause of the increasing inaccuracy in the Finapres was the development of peripheral vasoconstriction. Despite use of the usual methods of temperature conservation, the large exposure of the patient undergoing corrective thoraco-abdominal spinal surgery results in considerable heat loss. Vasoconstriction, due to increased autonomic nervous system activity, has been shown to cause a pressure reduction in finger arteries,

Table 2. Standard deviation of differences between the noninvasive blood pressure monitors and the arterial line for systolic, diastolic and mean pressures for all patients (mmHg). The clinically acceptable standard deviation of differences are indicated by bold figures.

	New Finapres			Old Finapres			Colin		
	Systolic	Diastolic	Mean arterial pressure	Systolic	Diastolic	Mean arterial pressure	Systolic	Diastolic	Mean arterial pressure
Patient 1	4.8	4.19	3.86	6.95	8.46	7.09	20.43	13.58	18.24
Patient 2	6.41	7.21	7.08	8.08	8.39	8.31	8.56	7.97	7.42
Patient 3	7.98	5.62	6.1	7.85	5.34	5.54	11.65	12.43	14.14
Patient 4	5.05	4.68	4.72	5.87	5.6	5.67	26.47	20.2	25.36
Patient 5	6.29	5.2	4.95	6.01	4.21	4.25	12.43	13.42	16.78
Patient 6	4.94	4.41	5.16	5.08	3.91	4.93	11.72	10.87	11.7
Patient 7	7.63	4.29	4.38	8.96	5.84	6.67	18.74	14.85	17.4
Patient 8	7.28	12.68	11.12	6.84	9.16	8.68	12.88	9.18	11.93
Patient 9	5.42	3.47	3.63	5.73	4.41	4.75	7.23	6.34	6.29
Patient 10	7.5	7.26	7.23	7.73	7.8	7.52	9.71	9.31	10.13
Patient 11	5.04	4.51	3.94	6.04	5.05	4.96	25.21	11.32	17.76
Patient 12	10.65	8.99	9.08	5.52	5.34	5.13	27.11	22.85	24.3
Patient 13	4.65	5.85	4.68	5.87	5.57	5.39	8.83	9.21	7.25
Patient 14	7.52	4.88	4.39	7.79	5.59	5.07	7.36	5.57	7.77
Patient 15	6.98	4.94	5.66	7.27	6.11	6.11	6.02	6.1	5.57

decreasing Finapres accuracy [17]. The development of finger venous engorgement in the dependent limb may also have confounded the data. This study did not evaluate the contribution to the time-related drift in the presence of variable vasoconstriction of the Finapres monitor. However, both Finapres devices performed with accuracy in the same patients (Table 2) intimating that patient-related factors influenced the acquired data.

The new and old devices performed with consistent accuracy across the arterial line mean arterial pressure ranges, although there were few readings in the < 60 mmHg range. The tendency of the Finapres to overestimate diastolic and mean pressures occurred independently of actual (arterial line) mean arterial pressure. This is consistent with the findings of Aitken and colleagues [8] who emphasised caution when interpreting Finapres readings when the systolic blood pressure is low. The Colin was also less accurate in the lower blood pressure ranges, in keeping with previous studies [6, 15].

The contemporary role of the Finapres in clinical practice depends upon its acceptance as a more accurate and reliable alternative to oscillometric devices. Its present status does not make it an acceptable alternative to direct arterial pressure measurement, particularly in patients with low digital perfusion requiring continuous blood pressure monitoring. Further advances in Finapres cuff and transducer technology must improve its accuracy under these conditions.

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Forum

Light anaesthesia with propofol for paediatric MRI

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Summary

Anaesthetic techniques and monitoring equipment may interfere with the technical demands of magnetic resonance imaging. The purpose of this study was to evaluate the safety and efficacy of a light anaesthetic technique with intravenous propofol in nonintubated children. In 20 neuropaediatric patients sedation was induced with propofol 1 mg.kg⁻¹, followed by a continuous infusion titrated to produce adequate immobilisation. Oxygen, 4 l.min⁻¹, was administered by paediatric face mask. Respiratory rate, end-tidal carbon dioxide tension and oxygen saturation were continuously monitored. In 10 patients capillary blood gas tensions were determined 3 and 20 min after the procedure. Data are reported as mean (SD) and the mean (SD) total propofol dose was 5 (2) mg.kg⁻¹.h⁻¹. Oxygen saturation remained constantly higher than 96% in all patients. End-tidal carbon dioxide tension varied between 35 (7) mmHg 3 min after induction, and 41 (6) mmHg 30 min after the start of the procedure. End-tidal to capillary PCO₂ difference was 4 (3) mmHg. Within 20 min after the end of the procedure all patients were fit for dismissal to the ward. One imaging sequence out of 49 was repeated because of movement artefacts. In conclusion, intravenous propofol sedation appears to be a safe and reliable technique for paediatric sedation during magnetic resonance imaging.

Key words

Anaesthesia; paediatric.

Anaesthetics, intravenous; propofol.

Magnetic resonance imaging (MRI) is increasingly popular and several technical aspects of this technique have specific consequences for anaesthetic management [1]. The strong magnetic field, produced by the superconducting magnet, attracts all ferromagnetic materials and induces electrical currents in ECG leads, pulse oximeter cables and temperature probes. The high frequency radiowaves may provoke heat production in metals, and can cause burn injuries, especially in small children.

MRI is extremely sensitive to motion artefacts. In contrast to computer tomography scanning, where images are completed consecutively, all MRI sequence images are obtained in the same time interval. Consequently, when movement occurs during MRI, the complete sequence needs to be repeated. Children may not be able to remain immobile for a sufficient period of time because of a frightening environment and periods of intense noise (up to 95 dB). A sedative or anaesthetic technique which is safe, reliable and compatible with the technical demands of MRI, is often required. The purpose of this investigation was to evaluate the efficacy and safety of a light anaesthetic technique based on the continuous intravenous administration of propofol in pediatric patients.

Methods

The study protocol was approved by the Ethics Committee of human investigation of the Catholic University of Leuven, and informed consent was obtained from all parents. Twenty neuropaediatric patients, with a mean age

of 38 months (1 month–12 years), in ASA classes 1 or 2 and scheduled for MRI, were studied. After premedication with oral atropine syrup 0.02 mg.kg⁻¹, sedation was induced with intravenous propofol 1 mg.kg⁻¹, immediately followed by a maintenance infusion at an initial infusion rate of 8 mg.kg⁻¹.h⁻¹. The infusion rate was subsequently titrated to maintain adequate immobilisation. When movement occurred, propofol 1 mg.kg⁻¹ was administered. The electrocardiogram (Siemens, Sirecust 404) was continuously monitored. Oxygenation and ventilation were evaluated with pulse oxymetry and end-tidal carbon dioxide tension measurement (Nellcor 1000). The monitoring equipment was placed outside the imaging room, and connected to the patient by extension sets. The respiratory gas samples were aspirated through a specially designed cannula with oral and nasal sampling ports. In 10 patients capillary blood gas tensions were determined 3 and 20 min after the start of the procedure. The duration of the procedure, mean total propofol dose, the requirement for supplementary bolus doses of propofol and the occurrence of moving artefacts were recorded. Data are presented as mean (SD).

Results

The procedures lasted 45 (6) min. The initial propofol infusion rate of 8 mg.kg⁻¹.h⁻¹ was decreased to 6 mg.kg⁻¹.h⁻¹ after 6 (3) min and the mean total propofol dose was 5 (2) mg.kg⁻¹.h⁻¹. In seven patients, additional propofol bolus administration was necessary to maintain

Table 1. SaO_2 and $\text{PE}'\text{CO}_2$ at 3, 10, 20 and 30 min following start of propofol infusion in children undergoing MRI. Values are expressed as mean (SEM).

	Minutes after start of propofol			
	3	10	20	30
SaO_2 (%)	99 (0.22)	99 (0.18)	99 (0.27)	99 (0.25)
$\text{PE}'\text{CO}_2$ (kPa)	4.65 (0.20)	4.97 (0.21)	5.05 (0.23)	5.45 (0.25)

Table 2. Age of the 20 patients (months).

(1) 1	(11) 35
(2) 5	(12) 38
(3) 9	(13) 39
(4) 11	(14) 40
(5) 14	(15) 40
(6) 14	(16) 42
(7) 18	(17) 44
(8) 24	(18) 57
(9) 31	(19) 120
(10) 34	(20) 144

adequate immobilisation. Out of 49 imaging sequences one had to be repeated because of motion artefacts. Oxygen saturation and end-tidal PCO_2 ($n = 20$) are summarised in Table 1. The mean end-tidal to capillary PCO_2 difference ($n = 10$) was 0.57 (0.09) kPa.

Oxygenation, ventilation and haemodynamic parameters remained within safe limits. All patients had recovered sufficiently to return to the ward within 20 min after the end of propofol administration. The ages of the patients are listed in Table 2.

Discussion

General anaesthesia with tracheal intubation would be unnecessary if there was a reliable and safe alternative, but while several techniques have been advocated for paediatric MRI [2], most of them have major disadvantages. The use of inhalation anaesthetics may be inappropriate if an increased intracranial pressure cannot be excluded [3]; the use of ketamine is contraindicated when intracranial pathology is present [4], and the duration and intensity of sedation with barbiturates or benzodiazepines is unpredictable [5]. Therefore, a total intravenous sedation technique using propofol is proposed. Propofol has a suitable pharmacokinetic profile [6], acceptable cardiorespiratory side effects [6] in this setting, and decreases cerebral blood flow and metabolism [7]. The dose of propofol required for immobilisation in this study was higher than that previously reported for sedation in adults. However, compared to adults, higher propofol dose requirements have been reported for general anaesthesia in children [8, 9], a finding that may be the result of both pharmacokinetic and pharmacodynamic differences. Pulse

oximeter function was only intermittently impaired by the dynamic magnetic field.

The small difference from capillary PCO_2 values indicates that continuous measurement of the end-tidal PCO_2 by means of a nasal cannula [10] is a reliable technique with which to monitor respiratory function in lightly anaesthetised children. Since the PCO_2 appears to increase after 30 min, respiratory depression cannot be excluded when longer sedation periods are necessary.

In conclusion, the reported technique appears to meet the demands of safety and has the advantage of simplicity, rapid induction and fast recovery. However, careful attention, extensive monitoring and the continuous presence of an experienced anaesthesiologist in the imaging room close to the patient is mandatory.

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Dermal patch anaesthesia for venous cannulation with 10% lignocaine gel containing glycyrrhetinic acid monohemiphthalate disodium as an absorption promoter

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Summary

The clinical efficacy of transdermal 10% lignocaine gel mixture containing 3% w/w glycyrrhetinic acid monohemiphthalate disodium as an absorption promoter was evaluated at venous cannulation in 17 paediatric (6–11 years) and 17 adult (29–65 years) patients. After about 60 min of occlusive application, the mean (SD) pinprick pain score was 0.7 (0.7) in the paediatric group, compared with 1.4 (1.3) in the adults ($p < 0.05$). Twenty-five patients (14 children and 11 adults) who had a pinprick score of 0 or 1 underwent venous cannulation without intradermal local anaesthetic. The mean (SD) pain scores at venepuncture showed no significant differences between children and adults.

Key Words

Anaesthetics, local; lignocaine, transdermal.
Anaesthesia, paediatric; premedication.

Skin puncture usually provokes sharp pain, even when a fine gauge needle is used. Furthermore, local anaesthetic solutions often produce moderate pain initially on intradermal or subcutaneous injection [1]. A variety of chemical [2, 3] and physical [4] methods have been tested as delivery systems for the transdermal infiltration of local anaesthetics. Perhaps the best known preparation currently available (not in Japan) is EMLA cream which was developed in the 1980s in Scandinavia [5] and is widely used in children before venepuncture [6–8].

Transdermal therapeutic systems have recently attracted a great deal of attention and new absorption promoters without adverse effects have been introduced. We planned to assess the usefulness of one of these for transdermal infiltration of lignocaine. After preliminary studies [9, 10], a 10% lignocaine gel mixture containing 3% w/w glycyrrhetinic acid monohemiphthalate disodium (GAMHPh) as an absorption promoter was developed. A recent double-blind study showed that this combination was more effective in alleviating pinprick pain and venous cannulation than lignocaine gel alone [11]. The present study examines possible clinical benefits of 3% GAMHPh–10% lignocaine gel mixture for the reduction of pain at venous cannulation in adults and in children.

Methods

With approval from the Institutional Ethics Committee on Human Research, we studied 17 adults (10 male, median age 43 years, range 29–65 years) and 17 children (9 male, median age 8 years, range 6–11 years), who conformed to ASA grades 1 or 2 and who were scheduled for elective surgery. Patients who had neurological disorders, or had been taking analgesics or sedatives were not studied. On the day before surgery, the effects of 3% GAMHPh–10% lignocaine gel were explained to the adult patients and to

the parents of the children. Informed consent was obtained from all subjects.

Preliminary studies indicated that an interval of 60 min was required for the treated skin to become anaesthetised [10, 12]. A dermal patch containing the gel (Table 1) was applied on the ward by the anaesthetist-in-charge, about 60 min before the anticipated time of venous cannulation. Approximately 0.3 g of the gel, soaked in a round sponge with a diameter of 25 mm and a thickness of 1 mm, was applied over a suitable arm vein and was covered with an adhesive plastic film (Tegaderm). Thirty minutes after application of the patch intramuscular atropine (0.5 mg) and hydroxyzine (50 mg) was administered to the adults; the children received atropine (0.2–0.3 mg) and hydroxyzine (20–30 mg).

The patch was removed in the operating room and the skin wiped dry of the gel. Adverse local reactions and the analgesic effect were evaluated by an experienced anaesthetist who was not involved in administering the patches. Patients were asked about any local irritation, particularly burning pain, caused by the gel or dressing. The treated area was then inspected for the presence of pallor, erythema, oedema or any other local reaction. The analgesic effect was assessed as follows: (1) The treated skin area was first pricked five times with a disposable 26-gauge sterile needle to determine the presence or absence of pain sensation. A pain score was obtained from each patient, by noting the number of painful pinpricks. In patients with a pain score of 0 or 1, a 20-gauge venous cannula was then inserted into the vein in the treated skin area without additional local anesthetic. Pain on cannulation was graded by the patients on a scale of 5, where 0 = no pain, 1 = little pain, 2 = moderate pain, 3 = strong pain, and 4 = extreme pain.

Values were expressed as means (SD). Statistical analysis was performed using the Wilcoxon *t*-test for scored values and the Student's *t*-test for the others. Values of $p < 0.05$ were considered significant.

Table 1. Composition of 10% lignocaine gel mixture with 3% w/w glycyrrhetinic acid monohephtalate disodium (GAMHPh).

Local anaesthetic lignocaine base	500 mg
Solvents propylene glycol	500 mg
ethanol	1500 mg
Absorption promoter glycyrrhetinic acid monohephtalate disodium (GAMHPh)	150 mg
Thickener Hibis Wako 104	50 mg
Neutraliser di-isopropanolamine	55 mg

Add sterilized pure water to make 5.0 g.

Results

The mean (SD) patch application time was 59 (7) min for the adults and 60 (6) for the children.

The mean (SD) pinprick pain score of 1.4 (1.3) in the adults was significantly higher than that of 0.7 (0.7) in the children. Twenty-five (74%) of the 34 patients had a pinprick score of 0 or 1; 11 (65%) in the adults and 14 (82%) in the children. The mean (SD) pain scores associated with subsequent venous cannulation were not significantly different; 2.1 (1.1) and 1.8 (0.9) for adults and children respectively.

In no case was it necessary to remove the local anaesthetic patch because of local irritation. Short-lived, self-limiting local erythema was observed in six patients (5 adults, 1 child) after removal of the patch.

Discussion

A series of absorption promoters were examined in an animal study [9] and the hydrophilic GAMHPh chosen for the hydrophobic lignocaine base in a concentration of 3% w/w GAMHPh is a derivative of glycyrrhetinic acid, which is contained in a variety of toiletries and is reported to have anti-inflammatory [13] and anti-allergic [14] effects.

It is the un-ionised (base) form of a local anaesthetic which chemically penetrates and diffuses into the tissues after transdermal administration, whereas the ionised form blocks the transmission of impulses in the nerve. The hydrochloride salt of an amino-amide local anaesthetic is water soluble and stable and the presence of water in such formulations enhances skin penetration. However, the concentration of the uncharged form depends on the pKa, so that only a portion of the local anaesthetic is in a form suitable for tissue penetration. Consequently, to be effective topically the local anaesthetic must be used in high concentrations [2]. Since local anaesthetic bases are freely soluble in various alcoholic solvents, it is possible to obtain a high concentration of the base by dissolving it in, for example, ethanol, as in local anesthetic sprays. As such preparations are nonaqueous they have poor penetrative properties if applied to intact skin.

It is possible to combine a high concentration of a local anaesthetic base with a high water content by using oil-in-water emulsions [3]. The eutectic mixture of lignocaine and prilocaine (unfortunately not available in Japan) has made it possible to increase the concentration of local anaesthetic in the emulsion droplets fourfold, while maintaining a low total concentration of local anaesthetic [5].

As an alternative we decided to study the feasibility of using absorption promoters already in use for transdermal therapeutic systems. For hydrophobic drugs, such as lignocaine, hydrophilic absorption promoters have been shown

to be generally more effective than hydrophobic compounds [9]. GAMHPh, which had been used for promotion of nasal absorption of insulin [15], was ultimately chosen as an absorption promoter for the gel after it had been shown to enhance the transdermal absorption of lignocaine in animals [9].

Following the findings of a subsequent investigation in man, the concentration of lignocaine in the gel mixture was increased from 2% to 10%, shortening the application time to 60 min [10, 12]. A double-blind study of transdermal 10% lignocaine gel, with and without 3% GAMHPh for pain reduction of venous cannulation, confirmed that the analgesic effect of the former was enhanced [11]. The 3% GAMHPh-10% lignocaine gel seems to have a similar analgesic effect and application time as EMLA cream [6-8] and a recent comparative study suggests this gel may be as effective as EMLA cream in producing local skin analgesia to pinpricks (unpublished observations).

In the present study the children evaluated the analgesic effects of the gel themselves. Children in this age group are thought to be able to understand the meaning of medical procedures and indicate the severity of pain [16]. We had expected that the gel would be more effective in children than adults because the greater thickness of the adult stratum corneum has been considered a major barrier to the transdermal absorption of drugs. The pain score for pinprick was significantly lower in the children than in the adults but, surprisingly, the pain score at venous cannulation was almost the same in the two groups. Soliman and colleagues observed that children aged 7-12 years consistently rated the pain and discomfort of venepuncture higher than either an anaesthetist or an observer; they emphasised the importance of the emotional component of the pain which children expect to occur with needles and venepuncture [17]. Since children can learn to be afraid of injections from a very early age, the routine use of local anaesthetic gels or creams can have very positive benefits extending beyond the immediate procedure.

Transient local erythema was observed in six of the 34 patients and was the only adverse local reaction. With EMLA dermal patches, similar transient local reactions have been reported [6-8]. Methaemoglobinaemia, due to the absorption of prilocaine from the EMLA cream, is of concern only in infants [18, 19].

A 60 min occlusive application of 3% GAMHPh-10% lignocaine gel is useful for reducing the pain of venous cannulation in both children and adults although further improvements are necessary in order to achieve ideal conditions.

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Shivering and pulse oximeter function

I was interested to read the recent editorial by Dr Crossley on peri-operative shivering (*Anaesthesia* 1992; **47**: 193–5).

One important area that deserves mention is the effect that shivering has on the function of pulse oximeters. These are widely used for patient monitoring in the recovery room where shivering, which has an inherent frequency of between 4 Hz and 8 Hz [1], is commonly observed. We investigated the effect of motion artefact on the function of four pulse oximeters and finger probes in normal volunteers [2] using frequencies of vibration between 4 Hz and 8 Hz to simulate the motion artefact produced by shivering. We found that all of the oximeters investigated were adversely affected by motion artefact producing spurious decreases in the displayed SpO_2 , the values often falling from 97% to 89–90% on starting the vibration sequences and persisting until the motion was stopped. We also noted that the displayed pulse rate was often incorrect.

Shivering may be a contributory factor in exacerbating postoperative hypoxaemia, but anaesthetists should be

aware that spuriously low displayed SpO_2 may be produced by motion artefact alone. Also some pulse oximeters are more prone to suffer from this problem than others. Motion artefact due to shivering may be reduced by the use of ear probes, and by adopting measures aimed at reducing postoperative shivering.

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Gastro-oesophageal reflux and hiccough on induction of anaesthesia

In a previous study from this Department, two patients had gastro-oesophageal reflux after hiccoughs at induction of anaesthesia [1]. Acid reflux occurs in patients with chronic hiccough, although it is not known if one causes the other. We wished to investigate whether reflux was inevitable after hiccough at induction of anaesthesia.

The study was approved by the Hospital Ethics Committee. Twenty-four adult patients, ASA 1 or 2, undergoing routine dental surgery were asked for informed consent. Patients were not studied if obese, if they had symptoms of gastro-oesophageal reflux, if they were assessed to have a difficult airway, or if they were taking drugs affecting gastrointestinal motility. pH was measured with an oesophageal electrode connected to a Digitrapp (Synectics Medical). Details of the apparatus, calibration and method of placement have been described [1,2]. The electrode was swallowed by the awake subject into the stomach, and the gastric pH measured. The electrode was then gradually withdrawn into the lower oesophagus, 5 cm above the gastro-oesophageal junction, which was identified by a sudden rise in pH.

The patient lay supine and anaesthesia was induced by a second anaesthetist with methohexitone 1 mg.kg⁻¹. Oesophageal pH was recorded continuously and the occurrence of hiccough noted. Anaesthesia was continued with inhalation of oxygen, nitrous oxide and enflurane by face mask. If reflux occurred, the electrode was withdrawn slowly, after deep anaesthesia was established, to measure the upper extent of the reflux. The study was then complete.

Eleven of the 24 patients gave consent, but four of the 11 were unable to swallow the electrode. The results are shown in Table 1. The mean intragastric pH of the seven patients studied was 1.97 (range 1.6–2.4), and the mean oesophageal pH was 6.6 (range 5.6–7.3). Three of the seven patients hiccoughed and all then had acidic lower oesophageal contents: pH 2.2, 2.0 and 2.4. The hiccoughs and acidification of the lower oesophagus occurred simultaneously. We could not define the upper extent of acidification reliably.

Only seven patients could be studied, but the three who hiccoughed all had reflux gastric contents. We cannot say if the one caused the other, but other evidence suggests it was

Table 1.

Patient	1	2	3	4	5	6	7
Gastric pH	1.8	1.6	2.0	1.9	1.8	2.3	2.4
Oesophageal pH	7.2	7.0	7.3	6.3	5.6	7.0	6.4
Hiccough ?	No	No	No	No	Yes	Yes	Yes
Post-hiccough oesophageal pH	—	—	—	—	2.2	2.0	2.4

the hiccoughs that caused the reflux. Fisher and Mittal report that infusion of acid did not precipitate hiccoughs in a chronic sufferer, and that successful surgery of gastro-oesophageal reflux fails to prevent hiccoughs; this was also reported by Shay *et al.* [3,4]. Methohexitone is not known to have an effect on the competence of the lower oesophageal sphincter.

In the previous study, the two who hiccoughed were also the only patients who refluxed. It seems likely that reflux is inevitable after hiccough, although this does not necessarily mean that regurgitation occurs or that patients are at risk of aspiration.

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Laryngeal mask: a more secure airway than intubation?

A 16-year-old boy presented for an emergency tracheostomy 36 h after elective removal of a thyroglossal cyst. For 2 h he had complained of increasing stridor and bilateral neck swelling of sudden onset due to haematoma, also involving the base of his tongue. His fasting time was uncertain and he was not premedicated. Monitoring was with ECG, noninvasive blood pressure cuff and pulse oximetry.

Following pre-oxygenation, thiopentone 350 mg was given cautiously and anaesthesia maintained with spontaneous ventilation using 100% oxygen and 3% halothane. The airway was difficult to maintain with a mask and Guedel airway. Intubation was attempted using a standard adult MacIntosh blade, but laryngoscopy was grade IV, no structures being visible beyond the base of the tongue, which was swollen with haematoma. At his initial operation he was noted to be a grade I laryngoscopy and tracheal intubation had been easy. Fibreoptic intubation

was also unsuccessful because no anatomical landmarks could be identified.

As a last resort, an Intavent size 4 laryngeal mask was inserted with ease, requiring only slight anterior displacement of the mandibular angle to secure a good clear airway.

Surgery proved to be technically difficult due to extensive haematoma in all tissue planes. When the trachea was identified, several attempts to site a size 8 tracheostomy tube were unsuccessful and eventually a smaller one had to be inserted. During this fairly prolonged procedure, the airway continued to be maintained with the laryngeal mask without difficulty. During recovery in the right lateral position, the patient vomited, indicating a full stomach at induction. Clear tracheobronchial suction indicated there had been no aspiration of gastric contents then or earlier.

This case indicates the potential usefulness of the laryngeal mask during a difficult tracheostomy. It secured

and maintained the airway when no other means were possible. During a conventional tracheostomy, when the tracheostomy tube is being inserted, or insertion attempted, the tracheal tube is withdrawn with the risk of the tube coming totally out of the larynx. In this case that problem was avoided during repeated attempts to insert the

tracheostomy tube. The laryngeal mask in this instance provided greater protection for the larynx than the only alternative available, namely a face mask.

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Use of the laryngeal mask airway in a patient with Edward's syndrome

We would like to report the use of the laryngeal mask airway in a patient with Edwards syndrome (Trisomy 18) and believe it is the first account of anaesthesia in this uncommon condition.

A 3-year-old, 10 kg female suffering from bilateral conductive hearing impairment, was scheduled for bilateral examination of her ears under general anaesthesia. She had been born at term after a normal pregnancy but it was quickly noticed that she grew at a slow rate and fed poorly. At age 2 years, she was on the third centile for weight and had the mental aptitude of a 6-9 month old infant. At this time, Edwards syndrome was diagnosed using chromosomal analysis.

On admission, the child was noticed to have a large occiput, micrognathia, high arched palate and low set ears; she had a short shield-like sternum, mildly hypotonic limbs and hypoplastic nails. She had a grade two systolic murmur which had been diagnosed as a clinically insignificant ventriculoseptal defect but which was regularly reassessed

by a paediatric cardiologist. She had had no previous anaesthetics, was taking no medications and had no known allergies. She was starved for 6 h and was premedicated with oral atropine and trimeprazine. Topical anaesthetic cream had been applied to the hands and the patient was accompanied into the anaesthetic room by her mother.

An oxygen saturation probe and an electrocardiogram were attached to the patient, who was anaesthetised with oxygen, nitrous oxide and halothane. The airway was easy to maintain and when the patient was sufficiently deep, a size 2 laryngeal mask airway was inserted. The operation (bilateral myringotomies and grommet insertions) progressed uneventfully and the patient made a good recovery, the laryngeal mask airway being removed when full reflexes had returned.

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Hoarseness and the use of the laryngeal mask

I read with interest the article by M. W. Jones *et al.* (*Anaesthesia* 1992; 47: 213-6) on hoarseness after tracheal intubation. Sadly it was extensive enough to pre-empt most of my own ideas about research in this field. Particularly impressive was the emphasis on qualitative assessment of the postoperative voice. From the list of factors investigated, however, there was one notable omission. Little work has been done on hoarseness following the use of the laryngeal mask, although the incidence has been quoted as being between 3.9% and 12% [1-3]. Only one study [3] has attempted to grade severity, which was merely equated with duration of symptoms. There would seem to be scope for applying Mr Jones's methods to postoperative vocal changes in patients in whom the laryngeal mask has been used, along with other factors such as the choice of inhalational agent or the use of intermittent positive pressure ventilation.

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Laryngeal mask cuff pressure and nitrous oxide

We note with interest the paper of Lumb and Wrigley on the effect of nitrous oxide on laryngeal mask cuff pressure (*Anaesthesia* 1992; 47: 320-3). We have measured the changes in laryngeal mask cuff volume during anaesthesia with nitrous oxide, oxygen and a volatile agent in 80 patients and found similar results.

Eighty ASA 1 and 2 patients, aged between 12 and 82 years, were studied. They all breathed oxygen, nitrous oxide and a volatile agent spontaneously via a laryngeal mask. Concentrations of nitrous oxide varied from 50% to 66%, and the length of anaesthesia from 10 to 115 min. Prior to insertion of the laryngeal mask, the cuff was

deflated as completely as possible by pressing the underside against a firm surface and at the same time aspirating with a syringe. Anaesthesia was then induced and the mask inserted in the normal manner. Once inserted, the cuff was inflated with 20 or 30 ml of air, for size 3 or 4 mask respectively, from a calibrated plastic syringe. At the end of the procedure the mask was deflated by attaching a 50 ml syringe to the cuff and withdrawing the plunger. It was then removed from the patient without detaching the syringe. Once out of the patient, the plunger was again withdrawn with the undersurface of the mask pressed against a firm surface to deflate the cuff maximally in the same way as

prior to insertion, keeping the syringe attached. Following this, the plunger was allowed to find its own level, and when all movement had ceased the position of the plunger against the scale was taken as the cuff volume at the end of the procedure. Gas proportions were maintained constant until the mask had been removed. Only then were the patients given 100% oxygen.

In the patients anaesthetised with 66% nitrous oxide ($n = 42$), cuff volume increased linearly with time and there was a correlation of 0.53 between length of time *in situ* and percentage increase in the cuff volume, which was highly significant ($p < 0.001$). With the lower concentration of nitrous oxide, although increases in cuff volume were seen, these were not significant.

Using 66% nitrous oxide the mean expansion after 1 h

was approximately 20%. As Lumb and Wrigley point out, this is much greater than the calculated expansion due to warming of the gases from room to body temperature, and could lead to problems when the laryngeal mask is used for long procedures. In our series there were two cases in which the quality of the airway deteriorated during anaesthesia; the simple act of removal of some of the gas from the cuff alleviated the problem.

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Hazards of supplementary oxygen

Further to the recent editorial (*Anaesthesia* 1991; 46: 905-6) and correspondence [1] on the hazards of supplementary oxygen, we wish to describe two patients who developed complications during administration of oxygen delivered under pressure.

The first, a 65-year-old man, had undergone an uneventful thoraco-abdominal oesophagectomy and his trachea was extubated in the ITU after 24 h of intermittent positive pressure ventilation; within 48 h he developed bilateral basal atelectasis and arterial hypoxaemia. He was treated with supplementary oxygen and 5 cmH₂O of CPAP via a mask. The second patient, a 71-year-old man, had been returned to the general ward following a cholecystectomy and choledochoduodenostomy. He was later transferred to the ITU with deteriorating lung function due to bronchopneumonia. After several days of IPPV, he was treated with oxygen and CPAP via a mask.

Within hours of commencing CPAP, both of these patients began to leak gas through their surgical drains. In addition, the first patient developed mediastinal and subcutaneous emphysema. The chest X ray of the second patient showed considerable free gas under the diaphragm, but there was minimal gas drainage from the T tube placed in the common bile duct. We assume that in both patients this complication represented swallowing of gas/oxygen under pressure, with subsequent leakage through the anastomosis, despite the presence of a patent nasogastric tube. The anastomotic leak in the second patient was confirmed by the passage of methylene blue dye from the

nasogastric tube into the abdominal drain, and a subsequent T tube cholangiogram showed the T tube to be misplaced within the peritoneal cavity.

It is our experience that the majority of patients with upper gastro-intestinal anastomoses treated with mask CPAP do not develop gas leaks and it is therefore unlikely that CPAP at low pressures can 'blow-open' a nonleaking anastomosis. We assume that these two patients already had anastomotic leaks and that CPAP made this apparent at an early stage. Furthermore, we are aware that some surgeons now test their anastomoses peroperatively by filling the bowel lumen with air under pressure; this would seem sensible, especially in patients who are at increased risk of postoperative respiratory dysfunction and who may require CPAP therapy. Finally, whilst we would state that all patients who have upper gastro-intestinal anastomoses should be denied respiratory support in the form of mask CPAP, we would now, in the presence of an established leak, elect to intubate the patient's trachea and provide CPAP via a tracheal tube.

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Fatal pulmonary embolus following Caesarean section

We read with interest the report by McHale, Tilhak and Robinson of fatal pulmonary embolism following spinal anaesthesia for Caesarean section (*Anaesthesia* 1991; 46: 128-30) in a patient with multiple risk factors for venous thromboembolism. To identify only three factors in her case, pregnancy, overweight and surgery, is to underestimate the danger to which she was exposed. For example, she underwent pelvic surgery lasting more than 30 min, which increased the risk [1], and she was not just a little overweight, she was grossly obese. Her height was not given but allowing for pregnancy, her estimated body mass index (kg.m^{-2}) must have been greater than 30. Did she have varicose veins? [1]; they are not mentioned as a risk factor.

The authors state there were no significant periods of

immobility and did not consider this a risk factor. However, trial of labour lasted 9 h and presumably the patient was in bed during this time. Surgery and the postoperative course added another 21 h of bed rest, making a total of 30 h of relative immobility in an 'at risk' patient. As it is suggested that a smaller volume of local anaesthetic solution could have been used for the subarachnoid block to give a less dense motor blockade, there has to be concern about muscle tone and the efficiency of the calf muscle pump during this critical period of immobility.

The large baby (4.08 kg) would have increased back pressure on the pelvic veins, and cephalopelvic disproportion exacerbated the problem. The spinal was performed in the right lateral position, but no mention is

made of surgical position. Was a wedge placed under the abdomen to relieve pressure on the inferior vena cava?

It is timely to remember the classic work of Hewson, who in 1771 showed that while static blood within an isolated venous segment remains fluid for some hours, the prior addition of clotting factors or activating substances to the blood is associated with rapid induction of clotting [2]. Thus, *in vivo* multiple risk factors potentiate the effect of the individual factor. With blood in a hypercoagulable state and venous return already impaired, it takes only a small added risk to initiate thrombosis, namely a small increase in pressure on the pelvic veins or a short period of immobility.

The number of direct maternal deaths in England and Wales from pulmonary embolism has fallen from 51 in 1970–72 to 24 1958–87, although this forms a greater percentage of the direct deaths (14.9 to 19.8%) [3]. Where multiple risk factors are already present in pregnancy, should prophylaxis be given for emergency section? The editorial on thromboembolism in the *British Medical Journal*, although directed at orthopaedic surgeons, is appropriate here, namely 'what is inescapable is the need for prophylaxis' [4].

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A reply

We thank Drs Wittmann and Wittmann for their interest in our report. We are delighted they agree with us that there may be a place for prophylactic anticoagulation in women undergoing Caesarean section, and we also feel elasticated stockings may be useful. We note they identified the same risk factors as us, namely obesity, pregnancy and surgery. We can confirm that the patient did not have varicose veins or, as far as we know, polycythaemia, thrombocythaemia, high plasma proteins, anti-thrombin III deficiency, reduced activity of vein wall plasminogen activator or any other predisposing factor to thrombosis.

We disagree that a labour of 9 h duration is a significant period of immobilisation in that the patient was not confined to bed until towards the end of this time. Indeed, in a primiparous woman a first stage of 9 h is entirely acceptable, even in those units where labour is 'actively' managed. All postnatal women are encouraged to mobilise as soon as possible after delivery, emphasising again the need for good analgesia. This was true for this patient, and she walked to the nursery and the bathroom during her first 24 h after Caesarean section. As is common practice, a wedge was placed under the patient's right hip for the duration of surgery.

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Difficulty in extubation

Professor Brock-Utne and colleagues (*Anaesthesia* 1992; 47: 229–30) described a case of difficult extubation caused by the common practice among anaesthetists of pulling off the pilot balloon and valve assembly from the tracheal tube in order to deflate the cuff. This method was commonly used because a syringe could not be located quickly. However, a syringe is not necessary in order to deflate the tracheal cuff. Every anaesthetist and recovery nurse should

have a pen to hand, the tip of which can be inserted into the valve assembly and used to depress the plunger allowing quick cuff deflation. There should therefore be no need to pull off the valve assembly in order to deflate the cuff in a hurry.

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Making up inotrope solutions

We would like to comment on M.J. Rooney's suggestion for a method of preparing inotropes for infusion via a 50 ml syringe and driver (*Anaesthesia* 1992; 47: 83). This is based on the assumption that it is desirable to arrange such infusions so that a 1 ml.h^{-1} infusion rate corresponds with a dose of $1 \mu\text{g.kg}^{-1}.\text{min}^{-1}$. His assertion, that this is a widespread practice, is certainly at variance with our experience: surely it is far more common to administer drugs in standard concentrations, inotropic agents included?

Notwithstanding this, the scheme he proposes is at best impractical and at worst potentially dangerous. Firstly, it cannot be applied to inotropes other than dopamine and dobutamine; the statement: 'This formula can be used with

any drug' may be literally true, but the volumes so derived for other inotropes verge on the ridiculous. Secondly, the method outlined is only applicable for patients of more than 70 kg; at lower weights, concentrations must be increased, defeating the supposed object of the exercise. Thirdly and most importantly, each infusion prepared is suitable for only one patient; combined with the necessity to vary concentrations for lower body weight patients, the potential for error in preparation and administration is obvious, particularly in the context of a busy intensive care unit and when such infusions are required rapidly and at short notice.

A safer and, we suspect, far more widely used approach, is to add a standard mass of drug to a standard volume of

diluent. In the case of dopamine and dobutamine 200 mg and 250 mg respectively in 50 ml. The required infusion rate can then be determined from the formula:

$$\text{Dose } (\mu\text{g.kg}^{-1}.\text{min}^{-1}) \text{ at } 1 \text{ ml.h}^{-1} = \frac{\text{Mass of drug (mg)}}{\text{Body weight} \times 3}$$

In fact the solution of this equation, for patients over 65 kg, is conveniently close to unity with dopamine and dobutamine, providing a useful additional check that the selected infusion rate is in the correct range. This method thus has several advantages: it avoids the problem of wastage cited by Dr Rooney; it is applicable to a far wider range of patients and drugs; it eliminates the potential for error inherent in systems where concentrations are varied from patient to patient and is simple both to prepare and use.

In conclusion, we would strongly advise against the scheme suggested by Dr Rooney, if only on the grounds of safety.

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A reply

I am delighted to reply to the letter by Dr Train and Dr Levitt.

I contacted by telephone 25 intensive care units around England. The practice of administering dobutamine such that an infusion of 1 ml.h^{-1} was equivalent to $1 \mu\text{g.kg}^{-1}.\text{min}^{-1}$ took place in four units. This would suggest an incidence of between 10% and 20%. In my original letter I only said 'many' units use this practice, although I agree it is far more common to use a fixed concentration. The units that do use a variable concentration appear to be satisfied with this method. Although a calculation is needed when preparing the syringe, thereafter conversion of the infusion rate from ml.h^{-1} to $\mu\text{g.kg}^{-1}.\text{min}^{-1}$ does not need a calculator. With a simulated population of patients (mean weight 70 kg, SD 14) each having one syringe of dobutamine made up and discarding any left over drug, then about 30% of the drug is wasted. If such a unit in a year consumed dobutamine worth £30 000, then they may have been able

to save £10 000. My suggestion for these ICUs is to use the formula I suggested, using the full contents of the vial of dobutamine but making it up to a calculated volume dependent on the weight of the patient.

I am thus puzzled by the comments by Dr Train and Dr Levitt. The units that do vary the concentration according to the patient's weight would not continue using this method if they found it was impractical or dangerous. Although there is potential for error when making up the solution, there is equally potential for error when converting from ml.h^{-1} to $\mu\text{g.kg}^{-1}.\text{min}^{-1}$ with a calculator. The 28% of units that I contacted that use a 1 mg.ml^{-1} basic concentration, but occasionally double or quadruple it with some patients, may also be prone to a labelling error. In practice, it does not appear to be a problem.

Drs Train and Levitt argue that my method cannot be used for patients weighing less than 70 kg. In the final paragraph of my original letter I describe the example of making up 250 mg of dobutamine for a 50 kg patient. The final volume needed would be 83 ml. As one cannot get 83 ml into a 50 ml syringe, the answer is to make it up to 41.5 mls and call it 'double strength'. Multiplying the infusion rate by two to give $\mu\text{g.kg}^{-1}.\text{min}^{-1}$ is still a convenient calculation and does not defeat the object of the exercise. The basic formula can be used with any drug and the volumes so derived can be impractical or impossible. Take noradrenaline as an example: 20 mg of noradrenaline base for a 70 kg patient would need to be made up to 4.8 ml. The neat solution takes up 20 ml. The answer is to make the 20 mg up to 38 ml thus producing a solution that $1 \text{ ml.h}^{-1} = 0.1 \mu\text{g.kg}^{-1}.\text{min}^{-1}$.

The Intensive Care Unit at Lincoln County Hospital in Lincolnshire used to use 3 mg.kg^{-1} into 50 ml for dopamine and dobutamine infusions. They changed over to the formula I describe without any difficulties. Wall charts displaying final volumes for each weight as a confirmation of the calculation appeared to help. It does not matter what method is used to make up inotropic solutions as long as the method is understood, there is consistency within that ICU and that expensive wastage is minimised. The criticisms levelled by Drs Train and Levitt are not justified. The answers to them are in my original letter.

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Persistent narrowing of an armoured tube

Many anaesthetists prefer to use armoured tubes, not only for patients scheduled for head and neck surgery, but also for long procedures performed in the prone position. Although this practice has been recommended in standard textbooks, its justification has been challenged in cases where the patient's head remains stationary and the operative field does not encroach upon the airway [1]. I would like to add some evidence to this statement by warning against a false feeling of safety which might occur whilst using armoured tubes.

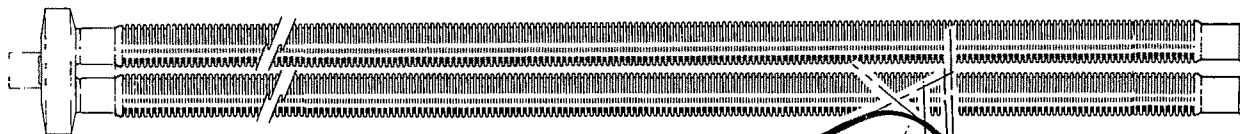
A 44-year-old woman with idiopathic scoliosis arrived on the intensive care unit for postoperative artificial ventilation after prolonged spinal surgery requiring massive transfusion and which led to some degree of hypothermia. At a routine attempt at tracheal suction, it was noted that a 16 FG suction catheter would not pass through the patient's size 7.5 mm internal diameter Mallinckrodt tracheal tube (Fig. 1). Bilateral air entry was confirmed, airway pressure was not abnormally elevated and there was

an adequate return of volume on the Servo ventilator. As the patient was awake with a normal temperature, able to clear her airway and haemodynamically stable with satisfactory oxygenation, it was decided to extubate her trachea. Fixed notching was observed at two sites on the tube, corresponding to the patient's teeth (Fig. 1). The same degree of narrowing of the tube could not be reproduced by firm kinking and therefore it was assumed that this shape of the tube was due to the patient biting on it. Although the observed reduction in diameter did not cause respiratory obstruction in our patient, it is disturbing that the tube did not resume its original diameter when the biting was relieved.

Re-inforced tubes are meant to reduce the degree of obstruction if inadvertent kinking occurs; however, by their virtue of being internally ridged, they lose their elasticity and once they are deformed they remain deformed. It is known that the use of a bite guard from a fiberoptic gastroscopist gives the best protection against biting on

The safer alternative

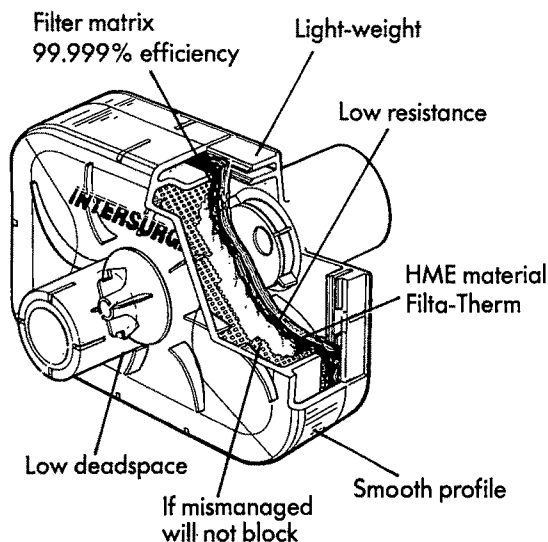
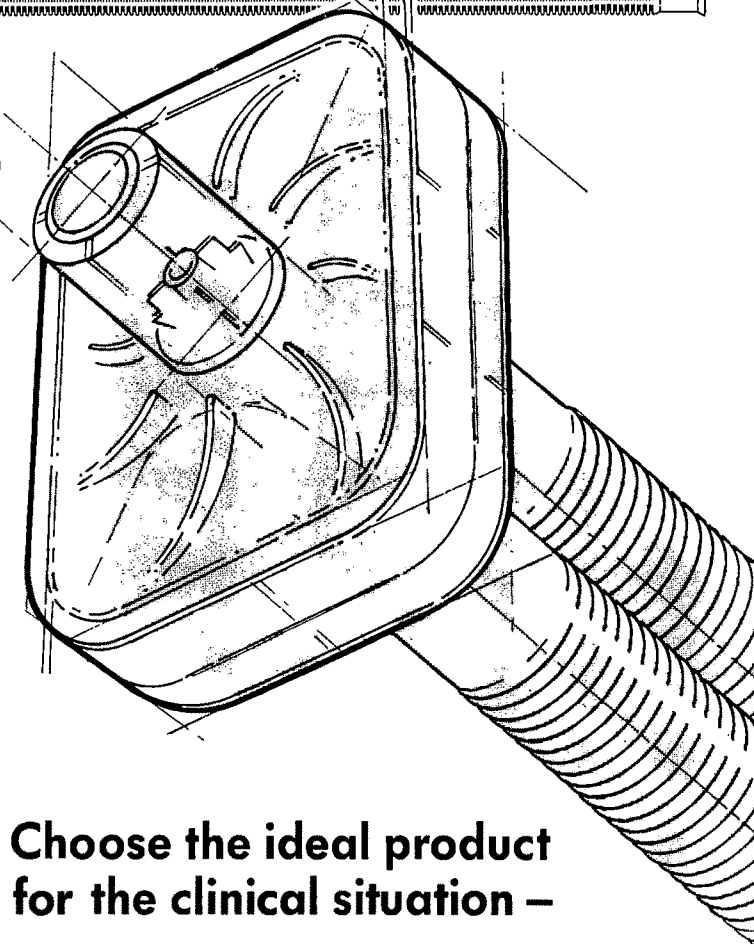
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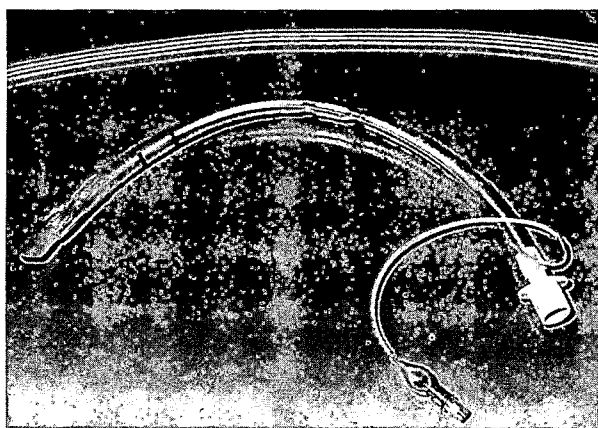


Fig. 1.

tubes. I hope this letter will alert anaesthetists to a potential hazard with tubes advocated as ideal for cases where, peroperative laryngoscopy may cause a problem.

AZ St. Jan Hospital,
Ruddershove 10,
8000 Brugge,
Belgium

P. MARTENS

Reference

- [1] WRIGHT PJ. Are armoured tracheal tubes really necessary for neuroanaesthesia? *Anaesthesia* 1986; **41**: 213.

A reply

I refer to the letter from Dr P. Martens regarding the partial occlusion of an armoured tracheal tube, which has been reported as being bitten by the patient.

The design of the Mallinckrodt reinforced tracheal tube helps to ensure an airway when the patient's neck is in an extended or flexed position. The reinforcing (armouring) prevents the tracheal tube from kinking in these extreme positions, whilst maintaining an adequate degree of flexibility. The tube is not designed to withstand the extremely high forces that can sometimes be exerted by the patient's teeth during recovery from a general anaesthetic. We would like to endorse Dr Marten's recommendations that a bite guard, from a fiberoptic gastroscope, or alternatively a Guedel airway would provide protection against partial occlusion should biting occur. We clearly state in all our support documents that 'the use of a reinforced tracheal tube does not exclude the use of a biteblock'.

We would like it to be noted that this response has been based upon the documented report of the incident, without being able to examine the product in question.

We would like to thank you for the opportunity to respond to Dr Marten's letter.

Mallinckrodt Medical,
D-5202 Hennef/Sieg 1,
Germany

J. WILLMOTT

Patient lifting systems

The accompanying photograph shows a 42-year-old man who had just spent the previous 30 min suspended on a lift above his Clinitron bed. He was at the time on synchronous intermittent mandatory ventilation and became extremely agitated, necessitating heavy sedation with propofol. The lift was used to allow maintenance to be carried out on the bed, without moving the patient to another bed, but it has no manual override and, if, as on this occasion, there was a failure of the electronic switch mechanism, there is no way to lower the patient back to the bed.

I understand that this is the first occasion that this has happened and I would urge anyone whose patient has to be raised on this lift to make sure a manual override device is available. Happily, this patient, who was in Intensive Care for 3 months following a ruptured oesophagus, is now making a full recovery.

Stobhill General Hospital,
Glasgow G21 3UW

R.L. HUGHES

A reply

We thank Dr Hughes for bringing this incident to our attention. We are in contact with the manufacturer of the



lifting system who is examining the components involved and the possibility of a manual override facility. This is to our knowledge the first such incident of its kind and we are assured the matter is receiving urgent attention.

SSI Medical Services Ltd,
Nottingham

P. STEVENSON

Epidural bupivacaine and diamorphine for postoperative analgesia

We were interested in the article by Drs Hobbs and Roberts and their experience of ward-based epidural infusions (*Anaesthesia* 1992; **47**: 58-62). In the Norwich area we have had an active postoperative pain service

managed by a dedicated sister for over 12 months. Our routine epidural infusions for postoperative analgesia consist of a combination of diamorphine 5 mg mixed in 0.125% bupivacaine. There are standard protocols for the

nurses to follow, with instructions for use of the infusion devices, an observation chart, and a contingency plan for any complications. These methods are employed for all surgical specialties, including thoracic, orthopaedic, general, gynaecological, and urological surgery. We have so far accumulated 287 patients.

We have encountered a number of problems. The commonest is discontinuation of the infusion by surgeons, predominantly juniors, because of systemic hypotension. Rather than increase intravenous fluids, the epidural infusion is seen as the cause of the hypotension. We now educate the junior surgeons in the management of epidural infusions. Epidural catheters are often difficult to place accurately, and we have found that up to 10% of our infusions fail because of catheter leaks or migration.

Although no patient has experienced severe pain, there have been 19 in whom the epidural has failed to produce adequate analgesia. These are usually transferred to a patient-controlled analgesia system. Any hospital wishing to start epidural infusions should have a reserve of PCA

machines to use in the event of either catheter problems or inadequate analgesia. We have had no instances of respiratory depression, and only three patients where the infusion had to be stopped because of nausea and vomiting.

A number of criteria are considered essential for satisfactory management: nurse education and training, which must be repeated frequently because of the high turnover of nursing staff; a designated nurse supervisor on each ward to bring any problems to the fore; a pain relief nurse dedicated each day to acute pain management; a well designed protocol to enable complete observations and awareness of rapidly developing complications; a standardised dosage regimen so that new anaesthetists may feel comfortable in starting this form of analgesia in the out-of-hours period.

Norfolk and Norwich Hospital,
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A. MORIARTY
B. JOHNSTON
G.E. PORTER

Respiratory depression after alfentanil

We present a patient who developed respiratory depression in the recovery room after intra-operative continuous infusion of alfentanil. This complication has already been described following fentanyl, pethidine morphine and sufentanil.

A 44-year-old, 72 kg man presented for partial gastrectomy. He had had no other previous illness and pre-operative evaluation was unremarkable. An hour before surgery he was premedicated with pethidine 75 mg and promethazine (50 mg). Monitoring included ECG, arterial blood pressure pulse oximeter and nerve stimulator. Anaesthesia was induced with thiopentone (5 mg.kg^{-1}), alfentanil ($60 \text{ } \mu\text{g.kg}^{-1}$) and atracurium (0.5 mg.kg^{-1}) intravenously. Maintenance was with 66% nitrous oxide, atracurium $10 \text{ } \mu\text{m.kg}^{-1}.\text{min}^{-1}$, and alfentanil $1.5 \text{ } \mu\text{g.kg}^{-1}.\text{min}^{-1}$ (total dose = 16 mg). The procedure lasted 150 min; atracurium and alfentanil infusions were discontinued 30 min before the end of surgery. The patient was completely awake 5 min after nitrous oxide was stopped, with the train-of-four showing complete recovery and a stable respiratory rate of $12 \text{ breath.min}^{-1}$. His trachea was extubated and no anticholinesterase was given.

In the recovery room, he received supplementary oxygen by a nasal cannula (3 l.min^{-1}). Twenty minutes after surgery (50 min after stopping the alfentanil infusion) the patient became apnoeic, unresponsive, rigid and cyanotic with constricted pupils. Naloxane $200 \text{ } \mu\text{g}$ resulted in restoration of respiration and recovery of consciousness. He remained in the recovery room for 8 h without any other incident.

All opioids produce respiratory depression, but it is rare to see apnoea after a patient is completely awake. Recurrence or delayed respiratory depression has been explained in different ways. The end of surgical stimulus and drowsiness could increase opioid depressor effects. Respiratory acidosis can occur after tracheal extubation, increasing cerebral blood flow and alfentanil nonionized

fraction. All these factors can produce more opioid receptor stimulation [1]. Alfentanil, with a pK_a 6.5, will be less influenced by pH than other opioids. Respiratory depression has also been attributed to a second peak in plasma opioid level, as a result of redistribution from peripheral compartments, or rapid intestinal absorption of accumulated opioid [2]. Skeletal muscular mobility and return to normal temperature can also produce redistribution. Mahla *et al.* described two cases of respiratory depression after alfentanil infusion with similar doses to ours ($50 \text{ } \mu\text{g.kg}^{-1}$ plus $1.5 \text{ } \mu\text{g.kg}^{-1}.\text{min}^{-1}$) and at about the same time (45 min after the infusion) [3]. This second peak can also occur during surgery or some hours later [4].

It is important to remember that alfentanil can produce delayed respiratory depression, similar to other opioids, in a dose-dependent manner. The use of this drug, which is especially indicated in outpatient anaesthesia because of its short half-life, does not preclude monitoring in a recovery room.

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F. CASSINELLO
A. PERAL
A. ANDUEZA
I. MOURELLE

References

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Evaluation of the E25 for the Level 1 blood warmer

I have recently assessed an additional disposable warmer for the Level 1 series, a system for warming large, rapid fluid transfusions [1].

The new warmer (E25) differs from those previously available in that it is not a complete unit, but connects to a

conventional giving set and does not have an air eliminator; it has a conventional flow chamber. These differences mean that the warmer must be primed before insertion into the Level 1 warming unit. This is easily done and it must be ensured that the rigid heat exchanger, as

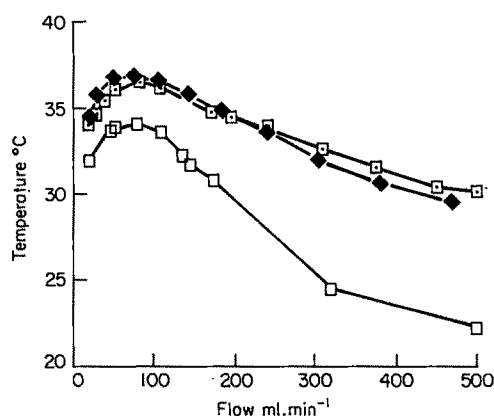


Fig. 1. Relationship between flow and issuing temperature for the E25 (◆), D50 (□), and Fenwall (□) warmers.

with all the disposable sets in this series, is not bent in any way during insertion into the sockets on the infusion stand. Bending of the rigid heat exchanger is easily avoided by sliding the upper socket upwards, completely clearing the top of the heat exchanger before sliding it back down to click firmly into position.

In a laboratory investigation, the E25, the D50 and the Fenwall warmers were compared. The Fenwall blood warmer was used in this evaluation as it is the alternative warmer in use in this department. Saline 0.9% cooled in a refrigerator overnight to a temperature of 5.0°C was passed through the D50 and the E25 disposables attached to the Model 250 Level 1 warmer at constant flows using an American Optical Roller pump. A similar experiment was performed with the Fenwall warmer. The flows could be varied between 20 ml.min⁻¹ and the 500 ml.min⁻¹. The temperature of the fluid entering the warmer and at the patient connector was measured using a Yellow Springs

Instrument Co. series 401 thermistor which had been calibrated against a National Physical Laboratories calibrated mercury in glass thermometer. The thermistor was held in the flow of saline in a way which did not obstruct the flow. The saline was collected in a measuring cylinder to calculate the flow. Each measurement was repeated three times, but there was no measurable difference between results.

The results are shown in Figure 1. There is little difference between the heating efficiency of the D50 and the new E25, both performing better than the Fenwall warmer. They both raised the temperature of the fluid to above 32°C at flows up to 300 ml.min⁻¹ and above 35°C at flows up to 180 ml.min⁻¹. The Fenwall only heated fluid to above 32°C for flows up to 140 ml.min⁻¹.

In 1974, Russell [2] laid down specifications for blood warmers, requiring blood to be warmed to a temperature greater than 32°C at flows up to 150 ml.min⁻¹. Since then, surgery and intravenous fluid requirements have increased so that these flows are no longer adequate. The Level 1 series was developed to meet this increased need, and with the range of disposables and hardware available, is appropriate for use wherever large fluid transfusions are given. The E25 is efficient, allows high flows, is easy to prime and set up. It overcomes the potential problem of the cost of the disposables, being approximately one third the cost of the D50, and should allow more general use of this valuable piece of equipment.

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J.M.B. SMALLMAN

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Intra-arterial prilocaine for ischaemia due to radial artery cannulation

Ischaemia and gangrene of the hand following radial arterial cannulation is a rare but well known complication [1]. The recommended treatment is immediate removal of the cannula and if signs of ischaemia do not disappear, a brachial plexus block [2] or even surgery [3].

A 54-year-old male patient presented in a state of shock due to a leaking abdominal aortic aneurysm. Radial artery cannulation was performed during the course of aneurysm repair using a 20 gauge cannula. A Doppler ultrasonic probe was used to aid in the localisation of the radial artery [4], and flow in the ulnar artery was tested at the same time. Postoperatively, despite having a manually measureable blood pressure of 100/55 mmHg at the brachial artery, the radial artery wave form was damped and perfusion of the hand was decreased; the Doppler signal was absent in the radial and ulnar arteries. The thumb, index and middle fingers were cyanotic and cold and the remaining fingers were blanched.

A mini intra-arterial regional block was performed by injecting 5 ml of 0.5% prilocaine (plain) through the radial artery cannula while the venous return was occluded by grasping the hand above the wrist for a duration of 2 min. This resulted in an immediate improvement in the circulation to the hand and the arterial trace re-appeared. Subsequently the patient's circulatory status improved and he did not require any further blocks. The same technique

has been used successfully in our unit in another instance of ischaemia following radial artery cannulation. The aetiology of ischaemia in both these cases was prolonged shock.

Intra-arterial injection of local anaesthetics is a safe technique and has been used as a method for providing regional anaesthesia, but its use for the relief of ischaemia due to radial artery cannulation has not been described previously. It is easy to perform and can be tried as a first line of treatment in such cases.

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V.J. SARMA

References

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Epidural infusion and neuropraxia

Epidural administration of saline was originally shown by Rice and Dabbs [1] to reduce the incidence of postdural puncture headache. Crawford, in 1972, reported a reduction in the incidence of such headache from 70% to 20% using 1–1.5 l of saline over 24 h [2]. This success, together with the original study of epidural compliance [3], has led us to regard the epidural space as being fairly compliant, and which can tolerate such an infusion of saline without causing a significant rise in pressure. We wish to report a case of neuropraxia occurring during an epidural infusion which questions the safety of this practice.

A 28-year-old, 70 kg, primiparous patient requested epidural analgesia during labour. The procedure was complicated by an accidental dural puncture at L₃₋₄ with a 16 G Tuohy needle. An epidural catheter was subsequently sited at L₂₋₃ and used to provide analgesia for a forceps delivery. An epidural saline infusion was commenced at 20 ml.h⁻¹ and 9 h later the patient complained of severe back pain associated with paraesthesia and numbness in the distribution of L₁ to S₄. Neurological examination failed to reveal any associated motor or autonomic abnormalities. The epidural infusion was immediately discontinued and the symptoms resolved over the next 3 h. A subsequent MRI scan was reported to illustrate narrowing of the spinal canal at the level of L₄.

This case illustrates that epidural compliance may be small and an infusion may then produce pressure effects and subsequent neuropraxia. The description of pain by the patient led us to stop the infusion; its continuation might have resulted in a more permanent neurological deficit. Pain on injection with any epidural procedure is an

important indication to cease injecting. It may represent an acute rise in epidural pressure or direct trauma. The practice of performing epidural saline infusions on a postoperative ward in the absence of an anaesthetist may result in delay in the termination of infusion if pain was to occur. If isotonic saline is to be used for the prophylaxis of postdural puncture headaches we would advocate the use of two bolus doses of 60 ml as an alternative to continuous infusion. This was first described by Craft in 1973 [4] and has the advantage that the anaesthetist is present throughout the duration of injection. This would allow one to terminate the injection at the first complaint of pain and thus limit any subsequent rise in pressure.

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S.D. WOODS
P. HODGSON
T. WALKER
D. HIGGINS

References

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Laparoscopy, surgical emphysema and ECG voltage

A 52-year-old, 60 kg male, presented for laparoscopic cholecystectomy. His past medical history was not significant and pre-operative clinical, haematological, biochemical examinations and electrocardiogram were normal. Routine anaesthesia consisted of oral pre-medication with diazepam 10 mg and induction with etomidate 18 mg and fentanyl 0.2 mg. His trachea was intubated with a 9 mm cuffed orotracheal tube following suxamethonium 75 mg. His lungs were ventilated with N₂O, O₂ and enflurane 0.5%. Muscle relaxation was achieved with a bolus dose of atracurium 25 mg followed by an infusion of 25 mg.h⁻¹. The Penlon Nuffield Ventilator (Anaesthesia Series 200) was used for intermittent positive pressure ventilation. Tidal volume was set at 600 ml, respiratory rate at 12 breath.min⁻¹. Monitoring consisted of continuous display of the ECG, noninvasive blood pressure, capnography and pulse oximetry. As the surgeons had to explore the common bile duct, duration of the operation was about 4.5 h.

Two hours after the start of the surgery, the ECG complexes were noticed to be of low voltage compared to those at the start of the operation and it was also noticed that both parotid regions of his neck and chest were

crepitant and swollen; surgical emphysema was diagnosed. The patient's peak inflation pressure remained the same, but oxygen saturation had decreased by 2–3% from 98–99%. The surgeons were informed and they immediately reduced the intra-abdominal pressure from 16 mmHg to 8 mmHg. As nitrous oxide might also have been contributing to the degree of cutaneous emphysema, it was decided to give 100% oxygen and anaesthesia maintained by increasing the enflurane concentration to 1.5% and introducing a propofol infusion 20–30 ml.h⁻¹. Pneumothorax was not suspected on the basis of stable haemodynamics and oxygen saturation. During the next 2 h the subcutaneous emphysema decreased. Chest X ray at the end of surgery showed subcutaneous emphysema, pneumomediastinum, but no pneumothorax. Postoperative recovery was uneventful.

This case illustrates that a diminution in ECG voltage, due to electrical insulation, could alert the anaesthetist of an ensuing surgical emphysema.

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12 Po Yan Street,
Hong Kong

N. YOGASAKARAN

Anaesthesia for transvenous insertion of an automatic implantable cardioverter-defibrillator

Carr and Whiteley state that the anaesthetic considerations for insertion of an automatic implantable cardioverter-defibrillator (AICD) as a formal surgical procedure are the same as for any major cardiac procedure (*Anaesthesia* 1991; 46: 737–40). The anaesthetic implications of transvenous

insertion of an AICD may be different. The following case illustrates some of the problems associated with providing anaesthesia for transvenous insertion of an AICD.

A 56-year-old, 80 kg male patient was scheduled for transvenous insertion of an AICD for repeated episodes of

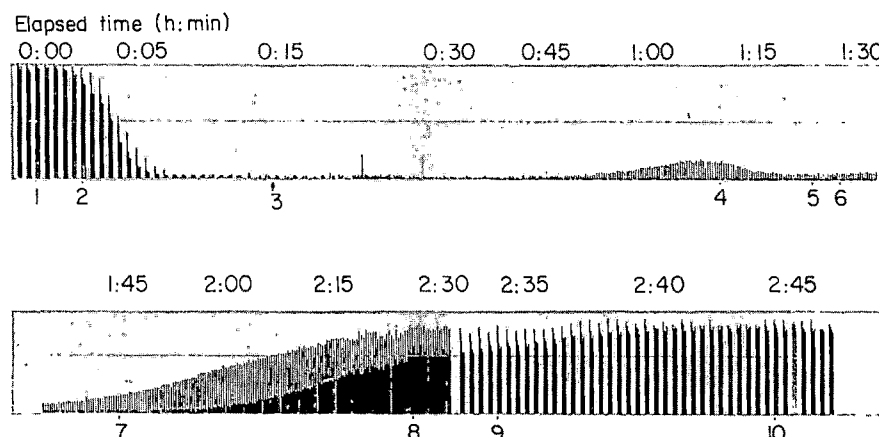


Fig. 1. (1) Intravenous atracurium 10 mg; (2) atracurium 10 mg; (3) infusion of atracurium 10 mg.h^{-1} started; (4) atracurium increased to 50 mg.h^{-1} ; (5) atracurium reduced to 10 mg.h^{-1} ; (6) atracurium reduced to 5 mg.h^{-1} ; (7) atracurium discontinued; (8) 75% TOF ratio reached; (9) intravenous neostigmine 1.25 mg glycopyrronium 0.25 mg administered; (10) 95% TOF ratio reached.

Recovery Index 25 min.

sustained ventricular tachycardia progressing to ventricular fibrillation. The patient suffered from dystrophia myotonica associated with respiratory muscle weakness (FEV_1 1.56 l, predicted 3.15; FVC 2.22 l, predicted 4.4; FEV_1/FVC ratio 70%; peak expiratory flow 150 l.min^{-1} , predicted 592). Arterial blood gases whilst breathing air were PO_2 7.9 kPa and PCO_2 5.9 kPa.

ECG, noninvasive blood pressure and oximeter monitoring were instituted. In the anaesthetic room, monitoring of the compound evoked electromyogram of the hypopharyngeal muscles in response to a train-of-four pattern of stimulation of the ulnar nerve, was also commenced (Datex Relaxograph). After pre-oxygenation, anaesthesia was induced with propofol 50 mg and fentanyl 100 μg , followed by an intravenous infusion of propofol at 5 mg.kg^{-1} supplemented by nitrous oxide in oxygen. Muscle relaxation for tracheal intubation was facilitated by two boluses of atracurium 10 mg and maintained with an intravenous infusion of atracurium at $0.125 \text{ mg.kg}^{-1}.\text{h}^{-1}$; a total of atracurium 50 mg was used during the 2 h procedure (Fig. 1).

Transvenous insertion of AICD involves three skin incisions, one in the left subclavian region for insertion of transvenous sensing/pacing wires, a vertical incision along the left anterior axillary border for insertion of the defibrillator patch and an abdominal incision for creation of a pocket in the rectus sheath to take the AICD pack. Once inserted, the device was tested three times by inducing

ventricular fibrillation; each time, after 10 s delay, a successful shock was administered. Ventricular fibrillation was associated with a decrease in end-tidal CO_2 from 5.4 kPa to $< 2.7 \text{ kPa}$.

Towards the end of the procedure, the atracurium infusion was switched off and neostigmine was administered when a train-of-four ratio of 75% had been reached. The propofol infusion discontinued 15 min before the end. Approximately 40 min after turning off the atracurium, the patient's trachea was extubated, at which time he had a measured tidal volume of 450–500 ml, with good cough and grip strength. The train-of-four ratio at this time was 95%. The patient subsequently received two further brief and uneventful anaesthetics for AICD threshold checks.

Transvenous insertion of an AICD is a relatively minor surgical procedure, for which a major cardiac anaesthetic is inappropriate. It can be argued that the ability to monitor blood pressure directly during induced fibrillation would have been an advantage. Whilst Carr and Whitely noted that the peripheral perfusion was inadequate for pulse oximetry during induced fibrillation, we found that the decrease in measured end-tidal CO_2 was also a useful indicator of the decreased cardiac output.

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J.N. CASHMAN
C. GARCIA-RODRIGUEZ
C. LAMOND

The 'Nonempty' empty bottle

Isoflurane (Abbott Laboratories) is supplied in bottles containing 100 ml of isoflurane costing £37.60 per bottle. At this price every drop counts and even a small wastage rate is financially significant. In the East Surrey Hospital isoflurane is used in the Ohmeda Tec 5 continuous flow vaporizer which has a key filler. The amount of fluid required to fully charge a vaporizer with dry wicks is 300 ml and the amount retained by the wick system is 75 ml [1]. In theory, using the key filler, it should be possible to deliver the entire contents of a bottle to the vaporizer without wastage. In practice, we have come up

against the 'nonempty' empty bottle syndrome whereby a small volume of isoflurane remains in the bottle which has been used to fill the vaporizer, even though the vaporizer is still not full to capacity. Usually this 'nonempty' empty bottle is simply capped and placed in a black plastic bag ready for collection and burial by the bottle bank.

To assess the wastage of isoflurane, we measured the residual volume in each of 94 such bottles which represented one month's supply of isoflurane. Vaporizers were filled under standard conditions and according to the key filler operating instructions [1] by nine operating depart-

ment assistants (ODAs). When a bottle was designated 'empty', the residual volume was measured using a quill and syringe. These measurements were performed by the two ODAs carrying out this study. Results showed an average residual volume of 3.22 ml per bottle (SD 1.40, range 0.9–6.3 ml, 95% confidence interval 2.93 to 3.51 ml). The residual volume was not significantly affected by the experience of the filler. The wastage of isoflurane in this hospital therefore cost approximately £114 per month.

Abbott have since informed us that they may shortly supply an alternative filler from Southmedic Inc of Canada. The new filler will they hope negate the 'nonempty' empty

bottle problem and will be a longer life product in general use.

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P.H. WITTMANN
F.W. WITTMANN
T. CONNOR
J. CONNOR

Reference

- [1] OHMEDA TEC 5 CONTINUOUS FLOW VAPORIZER. Operation and Maintenance Manual. September 1990.

Medical audit—failed intubation in obstetrics

The suggestions made by Dr Lockie and Dr Potter (*Anaesthesia*, 1992; 47: 273) deserve support; they underline similar points we made recently [1,2]. There is now evidence that the very high failed intubation rates that have been found in obstetrics can be much reduced, but efficient audit is essential to this strategy.

May we add another suggestion? We have approached Portex Ltd. about the possibility of their sponsoring an inter-hospitals intubation cup to be awarded each year to the hospital with the smallest number of failed intubations in, say, the last 1000 obstetric general anaesthetics. Friendly rivalry, like that roused by the rugby world cup, is a good way to focus interest. Maternal deaths are very rare, so it is all too easy to forget about them, but arguably they are the most important unsolved problem we have, and

reducing the number of failed intubations would certainly reduce mortality.

Portex have kindly said they would think about it; the support of a distinguished international journal, like *Anaesthesia*, would be valuable.

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R.S. CORMACK
F. CARLI

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'Humbug'

Recently a 91-year-old female suffering from paranoid schizophrenia was admitted to this hospital suffering from intermittent attacks of stridor and cyanosis. She was said to have been consuming mint humbugs. She gave no history herself and was uncooperative at examination, but an attack was witnessed by the ENT staff who suspected intermittent obstruction due to a foreign body. The attending ENT surgeon carried out a rigid bronchoscopy, anaesthesia being initially maintained with intravenous propofol, suxamethonium and apnoeic oxygenation. The surgeon had some difficulty passing the bronchoscope but once passed through the larynx no pathology was found and oxygenation was maintained using a standard insufflation technique. The bronchoscope was then

inadvertently withdrawn whilst the patient was still paralysed and the anaesthetist performed a direct laryngoscopy in order to intubate the patient. She was puzzled to find her view of the larynx obscured by a pinky-orange coloured mass. Extraction with Magill's forceps revealed a complete apricot, some 5 cm in diameter. Subsequent recovery was uneventful.

The surgeon suggested that the patient 'must have coughed it up' but the anaesthetic observation was that this was 'humbug'.

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G.L. MCAULIFFE

The Annual Conference of Linkmen of the Association of Anaesthetists of Great Britain and Ireland, 1991

The President, D. P. J. F. Baskett, welcomed 205 Linkmen to this meeting held in the International Conference Centre, Harrogate on the 11 September 1991. He began by noting the important role played by Linkmen in many aspects of the work of the Association. He emphasised that the Linkmen Conference was a forum for comments and criticism but, above all, for ideas for the future.

He drew attention to the magnificent venue offered by the Harrogate International Conference Centre. In particular, he praised the outstanding efforts of Dr R. S. Vaughan and Dr. F. L. Roberts in organising the biggest technical exhibition yet seen at an Association meeting, and urged Linkmen to visit it during their time at the meeting.

Independent practice

Dr. R. M. Weller (*Bristol*) reviewed matters relating to independent practice in the preceding 12 months. He noted that publication of the British Medical Association (BMA) publication entitled *Private Consultant Work: BMA Guidelines 1990* had been held up for 2 months while the anaesthetic section was being re-written with the help of the Association of Anaesthetists. The book was finally published in December 1990, and the guidelines indicated in this book were endorsed by the Private Practice Committee of the Association as being fair and reasonable. In April of this year an increase of 10.5% in the fee guidelines was recommended to members in keeping with the award of the Doctors and Dentists Review Body.

At present, all the provident societies, with one major exception, appear to be willing to reimburse professional fees based on the BMA/Association guidelines. The Association's Private Practice Committee has recommended that the BMA guidelines be used until March 31 1992. Dr Weller reported that a revision of this document is taking place at present and strong input from the Association has been given and accepted.

The British United Provident Association (BUPA) had failed to address the majority of the points raised by representatives of the Association in a series of arduous discussions throughout the year. Dr Weller paid particular tribute to the tenacity and determination shown by the Honorary Treasurer, Dr W. R. MacRae (*Edinburgh*) and the Immediate Past Honorary Secretary, Dr. W. L. M. Baird (*Glasgow*) in the course of these discussions. Such progress as had been made was, he suggested, due in great part to their efforts. Recently, BUPA had asked the Association to rank scale the anaesthetics listed in the benefit schedule. This, in effect was a Relative Value guide. Dr Weller had undertaken this task personally and explained to Linkmen how this had been achieved.

Finally, he reviewed areas where there was continuing debate concerning the need for fees. These included pre-operative assessment for day case surgery, the initiation and continued management of patient-controlled analgesic drug infusions and different approaches and indications for epidural anaesthesia and analgesia. In a show of hands there was a clear view expressed that the majority felt that these services were part of the anaesthetic management as a whole and should not attract extra fees. It was agreed to

ask the Private Practice Committee to keep these matters under review.

Discussion

Dr G. B. Rushman (*Southend*) encouraged members with an interest in private practice to form anaesthetic group practices. He advocated the many advantages of co-operative ventures of this nature based on his own experience and that of others. Dr R. L. Hargrove (*Westminster*) reminded Linkmen of his position as adviser to BUPA. He found his position difficult to sustain at times but had always believed that he had represented the best interests of his professional colleagues when giving advice. He noted that he and 12 colleagues had prepared a relative value guide in the past. This had been ignored and he expressed concern that the same thing may happen again. He also held the view that any attempt to 'load' anaesthetic fees on the basis of higher ASA gradings was unlikely to appeal to BUPA. Dr Weller thanked Dr Hargrove for his continued help.

Dr P. Coleman (*Jersey*) urged that any proposed fee structures should be kept simple. He noted that surgeons had done well at the expense of anaesthetists and suggested that it was time to redress the balance. D. K. K. Dutt (*Birmingham*) outlined local difficulties where trainee staff were being asked to anaesthetise private cases upon NHS lists, and asked for suggestions to manage this problem. The President offered clear and unequivocal advice that only consultants and accredited specialists should carry out this sort of work, and that the surgeons responsible should be contacted and asked to arrange their lists appropriately.

The Honorary Secretary, Dr. J. E. Charlton (*Newcastle upon Tyne*) read out a letter from Mr P. Garrad-Cole, Director, Strategic Services of BUPA. Mr Garrad-Cole had asked that Linkmen be informed of BUPA's position in the discussions concerning the levels of reimbursement of anaesthetic fees. After a brief, but pointed, discussion it was clear there was no support for Mr Garrad-Cole's views and Dr Charlton was given a mandate to inform him fully of the opinion of the Linkman Conference.

Dr A. P. Madden (*Bristol*) felt that members should continue to express their concern and anger about the failure of BUPA to reimburse fully anaesthetic fees. He suggested that as many members as possible should write to express their views. On another topic he suggested that it was the placement of the epidural catheter that was the additional service which should attract the fee and that the continued management was not as important in terms of remuneration. Dr Weller suggested that a percentage of the fee for an epidural as an independent procedure might be appropriate, and this was agreed by a large majority of those at the meeting.

Dr. D. P. Cartwright (*Derby*) noted that BUPA were capricious in their system of payments, particularly with regard to epidurals. He noted that the Association policy is that pre-operative assessment should form part of the overall anaesthetic fee. However, he felt strongly that pre-operative assessment in special cases should be reimbursed in exactly the same fashion as it would be for a surgeon or

physician carrying out a similar assessment. There was general agreement with this view.

Dr P. V. Scott (*Redditch*) expressed concern about the concept of a separate fee for epidural and patient-controlled analgesia. He suggested this may open the door for a reduction in fees if either of these methods of post-operative analgesia was not employed.

Dr J. S. Gibson (*Leeds*) drew the attention of the meeting to the increased length of time and the difficulty that can be experienced with anaesthesia for laparoscopic cholecystectomy. He felt that this procedure should attract an increased fee. Dr Weller felt that the time and number of difficulties experienced would decrease as the surgeons gained more experience with the technique, but promised to bring the matter up in discussions with the BMA representatives when considering the new guidelines. Dr Ann F. Naylor (*Basildon*) supported Dr Gibson's view. She also suggested that wider use of patient-controlled analgesia would attract financial support if its use could be shown to reduce the length of in-patient stay of patients.

There were further contributions from Dr D. J. Wilkinson (*St Bartholomew's*) and Dr P. M. Brodrick (*Mount Vernon*).

The role of the anaesthetist in the emergency service

The President, Dr P. J. F. Baskett, reviewed this recent publication of the Association. He praised the hard work by members of the working party who had prepared this report. It contains sections on anaesthesia and analgesia in the Accident and Emergency (A & E) Department, Resuscitation within the A & E Department, the role of the anaesthetist in major incidents and in the training of ambulance and paramedical staff. He drew attention to the extensive appendix containing a comprehensive list of equipment for mobile teams.

Discussion

Dr K. K. Dutt (*Birmingham*) enquired as to the correct site for emergency theatre work. He pointed out the problems caused by split site working and wondered whether there should always be a dedicated emergency theatre. In reply, Dr Baskett pointed out the advantages of making effective local arrangements.

Dr E. L. Lloyd (*Edinburgh*) felt that this was a valuable document, but considered there were some additions that could have been made. He believed that there could have been a section relating to the training of general practitioners and the lay public on resuscitation techniques. He asked for consideration to be given for the establishment of SHO posts to train general practitioners, especially if there was a BASICS scheme working in the area.

Dr R. L. Hargrove (*Westminster*) informed the meeting of his experiences using peripheral neural blockade as a most effective method of analgesia. The only difficulty he was aware of was that this made consequent assessment of neural function more difficult. Dr Anne J. Sutcliffe (*Birmingham*) suggested that the solution to this was to have the surgeons assess neural damage prior to the block being performed.

Dr Margaret L. Heath (*Lewisham*) felt that the need for radiology during resuscitation should be considered, and that an efficient solution might be for each resuscitation bay to be covered by a mobile machine. This would mean that main radiology departments need not remain open at night and problems with monitoring and care of patients in these circumstances would be reduced.

Dr P. V. Scott (*Redditch*) complimented the Association

on the excellence of the publication which he felt would be helpful to all concerned. He drew attention to the problems associated with the need to transport patients with an accompanying anaesthetist. This may have the effect of leaving the transmitting hospital without adequate levels of cover for other emergency duties. He also noted that delays in transfer for any reason could lead to adverse patient outcome.

Dr J. H. McClure (*Edinburgh*) wished clarification of the recommendations for cover in the section on resuscitation. He felt that these might be at variance with current guidelines from the College of Anaesthetists. In reply, Dr Baskett indicated that the intent of the recommendations was not to impose a solution but to bring levels of cover up to standard by gradual implementation of the guidelines. Dr J. W. Martin (*Burton on Trent*) felt that unrealistically large numbers of trainee staff were becoming involved with cover for many different aspects of anaesthetic practice. Recent agreements to reduce the number of hours worked by trainees may lead to great difficulties in providing appropriate cover.

Trainee hours of work

The Assistant Honorary Treasurer, Dr R. S. Vaughan (*Cardiff*), presented a summary of many of the current problems. The needs of training and service were inter-related and a balance had to be sought. It was not clear whether training would be extended consequent to the reduction in hours, nor was it clear how the posts of visiting registrars were to be filled. He noted that many trainees were unhappy with the salary reductions consequent to the decrease in hours. He felt that it had not been fully appreciated that the limitation in hours must take into account study leave, annual leave and absence for other reasons.

He emphasised that continuing medical education was vital for consultants as well as trainees. When taken in conjunction with the requirement to participate in management activities he foresaw grave problems with maintenance of service commitments. There were further problems with the performance of research. The concern of managers was for the continuation of the service and nothing else. The worry was that educational and research activities may become a secondary consideration.

Solutions to the problems anticipated with the reduction in trainee hours of work included maintenance of the status quo, which Dr Vaughan felt was totally unacceptable. Another possibility was a joint response from the Association and the College of Anaesthetists outlining all the difficulties. This should include a strong statement about the difficulties of providing cross cover because of the unique skills possessed by anaesthetists.

The potential difficulties caused by a reduction in the number of anaesthetists available for service work could be addressed in several ways. Dr Vaughan felt that the most fruitful line of development was by introducing the recommendations made by the Association's own report into the efficiency of theatre services which were endorsed in the Bevan Report on the same subject. Other alternatives were an increase in consultant numbers, an expansion of numbers of anaesthetists in Staff Grade posts and the introduction of nurse/technician anaesthesia. He concluded that the overriding concerns were for the maintenance of standards and the safety of the patient. Efforts should be concentrated on improving the efficiency of service provided by current staffing levels and that management should be involved at all stages of this process.

Discussion

Dr M. S. Pegg (*Royal Free Hospital*) noted that many trainee staff were concerned that changes in current work practices may effect adversely the quality of their life. The introduction of split weekends and partial shifts may lead to a reduction in hours but might also mean a restriction of liberty on two nights out of three rather than one. He felt that the solution lay in a rationalisation in workload. Dr G. B. Rushman (*Southend*) related his experience with the introduction of reduced hours of work for trainee staff. This had been achieved successfully without reduction in caseload, training or the quality of life for the trainees.

Dr P. Coleman (*Jersey*) informed delegates of the problems encountered in his practice. Jersey has a zero job growth policy which, he felt, made a mockery of the implementation of reduced hours of work for trainees. Dr D. J. Lightman (*West Essex*) was concerned that incorrect data with regard to many trainee posts was being supplied to regional manpower committees. In his own North East Thames Region he had anticipated problems and written to those concerned. The result of this informal survey had indicated that there may be serious problems with the grading of trainee posts. He outlined various ways of addressing these problems and urged that College Visitors pay the closest attention to trainee hours of work in future.

Dr A. P. Madden (*Bristol*) felt that the College of Anaesthetists response concerning trainee hours of work was inaccurate and illustrated this point of view with clear examples. It was also noted that there may be problems in the future with locum cover.

Honorary Secretary's report

Dr J. E. Charlton, Honorary Secretary, reviewed the work of the Association in the past year. He began by emphasising the enormous contributions of all members of Council, particularly that of committee chairmen and members of the various committees and working parties. He stressed that none of the work of the Association could occur without the smooth and efficient help provided by the Association's secretariat.

Once again the strength of interest in the affairs of the Association had been demonstrated by a strong field of candidates offering themselves for nomination to Council. Council had continued to be active on matters of concern to members. Comprehensive advice has been offered with regard to Consultant workload and the formulation of job plans. Council has submitted a strong and positive response to the report of the Department of Health working party on dental anaesthesia and has held discussions about the introduction of the new model Consent Form.

Working parties on departmental accommodation and secretarial assistance, workload for nonconsultant career grades, and hepatitis and HIV will be meeting in the forthcoming year. As in the past, the reports of the working parties will be published in the form of booklets with the aim of disseminating the conclusions of the working parties in an attractive and readable form. Other activities of Council were to be discussed during the Linkman Conference.

Council has agreed to provide financial support to the College of Anaesthetists to enable the purchase of suitable property. Details of the arrangements are to be found in the Annual Report. Dr Charlton felt sure he was expressing the view of Council and all members of the Association when he offered unswerving support to the ambitions of the College to become independent. Purchase of property by the College would move the goal of independence nearer

and permit the continued development of anaesthesia as a specialty.

The Association's education and research activities remained as strong as ever. An extremely successful Annual Scientific Meeting had been held at Manchester. The Postgraduate Study Day, organised jointly with the College of Anaesthetists, the Winter Scientific Meeting and the Group of Anaesthetists in Training Annual Scientific Meeting had enjoyed similar success. Dr Charlton drew attention to the large amount of hard work necessary to produce scientific programmes of such quality and to run meetings with such efficiency. He offered the thanks of the Association to those concerned.

A record 19 high quality applications were received for the Association Research Fellowship. After consideration of expert reports and interview of the main candidates Dr Jane Peutrell (*Bristol*) has been appointed to the Fellowship.

The Education and Research Committee had continued to adjudicate on applications for research and travel grants. Three grant requests had been approved as well as one travel grant. The committee had also approved an increase in the maximum sum that can be awarded in a travel grant to £750. The Baxter Healthcare Travelling Fellowship was awarded to Dr A. J. Lee (*Edinburgh*). Council is grateful to Baxter Healthcare for their continued support which is a valuable resource to the specialty. Three prizes were awarded in the clinical section of the Undergraduate Prize Essay. The seminar programme has continued in its enormously successful format and members were reminded of the need for early application to secure a place.

Dr Charlton drew attention to the reports of the chairman of committees and working parties in the Annual Report. He offered to provide further detail of any aspect of the work of the Association to any member. He stressed that the Association existed to respond to the needs and concerns of members and that continued dialogue ensured that these need and concerns were met. He thanked all those who had made his first year as Honorary Secretary so enjoyable.

Open forum 1

Training of Operating Department Assistants

Dr Jean Robson (*Guildford*) was anxious to know if the new National Vocational Qualification (NVQ) for Operating Department Assistants (ODAs) was to be introduced in the near future. She also expressed concern that hospital training schemes for ODAs may be affected by the introduction of NHS Trust status. In reply, Dr Margaret L. Heath (*Lewisham*), reviewed the new NVQ. She noted that the new scheme was not standard and could take the time and form necessary to train the individual. Approximately half the ODA schools were ready to start the new NVQ, whereas the remainder would continue to provide the City and Guilds qualification. She emphasised the need for anaesthetists to be involved in ODA training as many responsibilities in this matter were being devolved to local centres. She informed Linkmen of the formation of a communication circle to disseminate information concerning problems with the introduction of the new NVQ.

Anaesthetic machine checking procedures

Dr P. Smith (*Worksop*) asked for an opinion concerning the practice of ODAs checking anaesthetic machines rather than the anaesthetist. Dr. P. J. Bickford-Smith (*Bradford*) expressed an unequivocal view that the checking of anaesthetic machines was the sole responsibility of the anaesthe-

tist carrying out the operating list. This could be carried out in conjunction with an ODA but never without the direct involvement of the anaesthetist. He pointed out that the new anaesthesia work stations were of increasing complexity, and that it was likely that the age of anaesthetic machine stock will rise in the future. Both factors made it a matter of the greatest importance that checks were carried out by the anaesthetist.

This view was strongly supported by Dr W. R. MacRae (*Edinburgh*) and Dr R. L. Hargrove (*Westminster*). Dr Hargrove gave a clear opinion that should a negligence claim arise concerning anaesthetic equipment, that claim would be indefensible if a machine check had not been carried out by the anaesthetists concerned. Dr C. J. Barham (*East Grinstead*) reported an audit of machine checking in his Health District had revealed that a substantial number of machines were not checked by the anaesthetist prior to use.

Epidural versus general anaesthesia for delivery

Dr J. W. Martin (*Burton on Trent*) asked for an opinion concerning a view that it was unethical to give a general anaesthetic if an epidural had been used successfully for the management of labour. After discussion a clear view emerged that the views of the mother must be taken into account and that it was perfectly acceptable to administer general anaesthesia in these circumstances.

Seminars at 9 Bedford Square

Dr Agnes Leslie (*Birmingham*) enquired whether it would be possible for District General Hospitals to 'block book' a place for the Seminars at 9 Bedford Square. Her experience indicated that these were frequently oversubscribed and that she felt that those in the periphery may be at a disadvantage when booking. In reply the Honorary Secretary emphasised that all bookings were taken on a first-come, first-served basis, and that there was no prejudice or bias in the selection of applicants for the seminar programme. However, unsuccessful applicants were given the opportunity to remain on a waiting list for a repeat of the seminar.

Dr N. B. Williams (*Bristol*) suggested that it might be possible to tape record the seminars for wider dissemination. In reply, Dr Vaughan said that this had been considered in the past, but was thought to be unworkable because of the informal nature of the programmes and the lack of the visual element of the presentation.

Testing for HIV

Dr G. Nunn (*Wakefield*) enquired about current policy about testing patients for HIV. Dr Charlton re-iterated the Association view that testing was permissible without the patient's consent provided the test was obtained for the purpose of clinical management. He also drew attention to the working party, under the chairmanship of Dr J. Searle, which would consider this and other matters related to HIV and Hepatitis B.

Registration of ODAs

Dr Jean Robson (*Guildford*) returned to this subject and asked that members of the Association continue pressure for central registration of ODAs as proposed in the Bevan Report. In support, Dr Margaret L. Heath (*Lewisham*) described the extensive efforts that have been made in the past to achieve this goal. It was Dr Heath's opinion that registration of ODA qualifications will lead to professional

accountability, but that this obvious step forward was being resisted by the National Health Service Management Executive.

Anaesthesia on remote sites

Dr Luane J. Mackay (*Sunderland*) asked if the Association might give an opinion about the conduct of anaesthesia on remote sites and the facilities necessary. As an example this might be anaesthesia for electroconvulsive therapy. There was extensive discussion concerning this topic and it was emphasised that the facilities and levels of assistance should be no different from those necessary in any other anaesthetising location. Dr P. Morris (*Manchester*) stated that the College of Anaesthetists felt that no trainee should be asked to anaesthetise in these circumstances without supervision.

Audit

Dr Jennifer M. Eaton (*Bristol*) began by describing her approach to audit as personal and pragmatic. In a wide-ranging presentation she offered an opinion on how audit could be achieved best within the anaesthetic department. Audit should be carried out to improve current practice and permit the implementation of change. The audit process included reading the literature, writing papers, holding joint discussions with colleagues in other disciplines and discussing peri-operative deaths and critical incidents. She emphasised that the whole anaesthetic department must be involved and that the process should be kept simple.

Topic, criterion-based audit was potentially the best type of audit, but this may be difficult for anaesthetic departments. Alternatives include an audit of specific indices and an audit of workload. The latter might be achieved using the logbook system but there were clear problems in differentiating between audit and management when workload was being considered.

Dr Eaton was of the firm opinion that audit should be carried out within the working day, and that a rotating monthly hospital-wide meeting was a minimum. It was thought appropriate that some meetings be closed and that collaboration with colleagues from other disciplines be sought for others. In conclusion, she stressed that the quality of care provided was the standard by which the audit process must be judged.

Discussion

Dr P. V. Scott (*Redditch*) agreed that a hospital-wide audit day was essential. He informed Linkmen of experience in the assessment of outcome and suggested that definitions of outcome be agreed. Events occurring in the recovery area could be termed immediate. Those occurring within 48 hours of the anaesthetic could be termed delayed and those occurring up to 30 days postoperatively could be termed remote. He suggested that consideration be given to the use of nursing colleagues to detect and report on delayed outcome problems and that an interdisciplinary survey would be required for identification of remote problems. He proposed the use of both pre- and postoperative questionnaires with an additional survey of postoperative follow-up clinics to achieve this.

Dr R. S. Vaughan (*Cardiff*) suggested that a solution to problems of scheduling an audit could be found with the appointment of an Audit Officer. This had worked well in a situation where the audit activities of three separate hospitals had to be coordinated. The Audit Officer had been appointed by management and this had provided the

impetus and authority to cancel clinics and other activities which were necessary to permit the introduction of a standard audit day. Dr Margaret L. Heath (*Lewisham*) suggested that audit should be restricted to Tuesday, Wednesday and Thursday; pointing out that Bank Holidays already led to cancellation of lists on Mondays and Fridays. She also suggested that theatre maintenance could be co-ordinated with audit activity. Dr Jean Robson (*Guildford*) made the point that nursing audit should be linked similarly.

Dr M. I. Carter (*Luton & Dunstable*) listed the problems he had encountered as lead audit officer in his Health District; not the least of which were the variable recommendations of the various Colleges as to the amount of time to be spent engaging in audit activity. This varied from making no recommendation up to one notional half day per week. It was his belief that rolling audit was the correct way to carry out audit within the hospital. Dr D. Childs (*Chester*) raised the problems with the anaesthetic minimum data set. He was particularly concerned with the possibility of misinterpreting the American Society of Anesthesiologists (ASA) pre-operative risk scale at the extremes of age. Dr Eaton agreed with this view and noted that the use of the ASA scale could be extremely difficult. Dr Childs felt that it would be appropriate for the College of Anaesthetists Quality of Practice Committee to issue a standard.

Dr G. Nunn (*Wakefield*) was concerned that the definitions used in the report of the Confidential Enquiry into Peri-operative Deaths were sufficiently concise. In particular, there was a need for a term to describe operations being carried out between 24 hours and 15 days, that is to say a term between 'urgent' and 'scheduled'. Dr Childs suggested that 'nonscheduled' might be suitable. Dr Juliet E. Goodwin (*York*) suggested that the term 'necessary' would be better; she also made the point that audit would be more effective if it was a fully coordinated regional activity.

European relations

Dr W. R. MacRae introduced the subject with a review of current regulations with regard to basic medical and specialist qualifications. Basic medical qualifications are now harmonised, but specialist qualifications are not harmonised and the member countries of the European Community vary widely in the accreditation of specialists. Variations are found in the time spent in training posts prior to accreditation, which may vary from the 7 years found in Great Britain and Ireland to as little as 3 years. In addition, training and facilities are regularly inspected in Great Britain and Ireland whereas in some European countries inspection of training posts is rare.

Dr MacRae drew attention to the fact that there was free movement and full registration available in Great Britain to all European doctors. He suggested that it was likely that increasing numbers would avail themselves of opportunities to work in Great Britain and that this might lead to problems with specialist accreditation or even discrimination. An attempt was being made to sort out the potential and real problems through the European Boards. The aim was to assess all aspects of training and introduce examinations to permit assessment of comparability. The European Academy goes some way to meet these aims but was not yet a representative body. He noted that the European Diploma in Anaesthesiology was run by the European Academy. Future meetings would be held to merge the aims of the various European bodies concerned with training in anaesthesia. Dr MacRae believed that the

Association and British anaesthesia was well represented in this process but that there were no quick or easy solutions available.

Dr D. E. N. Evans (*Cardiff*) urged that the representatives should strive as hard as they could to bring the rest of Europe in line with Great Britain. He expressed concern at the discrepancies in training and suggested that a solution might be found by linking training with the European Diploma. In reply, Dr MacRae reviewed the efforts being made to increase training to a 5 year period. He noted resistance to these efforts which were based upon many factors not the least of which were financial problems and national pride. The President, Dr Baskett, noted that all national societies supported a five year training programme.

In response to a question from Dr T. D. Sharpe (*Belfast*) concerning the experience and training of European doctors, Dr MacRae outlined the steps necessary for agreement to change regulations in Europe. He noted that the European Academy had organised a voluntary inspection scheme to indicate to participants how matters might be improved. However, it had been noted that many European universities were resistant to the idea of an outside body conducting inspections as they viewed this as a threat to their own powers.

International relations

Dr S. M. Lyons (*Belfast*), the Chairman of the International Relations committee, reviewed the work of the committee. The principal aims of the committee were to improve practice and help the development of anaesthesia in other countries, particularly in the developing world and Africa. Dr Lyons noted the role of other societies and bodies and emphasised the need for improved coordination. The Association's contributions include the provision and funding of lecturers to courses organised by the World Federation of Societies of Anaesthesiologists (WFSA), placements of British anaesthetists in posts abroad and aiding the British Council in finding posts in Great Britain for overseas doctors. Other activities include collecting books and journals for overseas departments and the maintenance of a database on overseas experience at 9 Bedford Square.

Dr Lyons outlined a new initiative on the part of the Association to increase the number of lecturers participating in overseas courses. He had approached national, regional and specialist societies for contributions toward the cost of this exercise. The society would nominate the lecturer and contribute half the cost, the remainder being provided by the Association. The first lecturers under this scheme had already been nominated by the parent societies and Dr C. Monk (*Bristol*) would participate in the WFSA Moscow course sponsored by the Society of Anaesthetists of the South Western Region, and Dr I. Levack (*Aberdeen*) was to travel to East Africa under the auspices of the Scottish Society of Anaesthetists. Dr Lyons hoped to inaugurate a three year rolling programme of participation of this nature.

Dr G. Nunn (*Wakefield*) and Dr T. D. Sharpe (*Belfast*) expressed support for these activities. Dr Nunn suggested that members of the Association may care to convenant fees to support the activities of the International Relations committee. Dr Nunn also made an appeal for equipment, books and recent journals to be sent as urgent aid to Romania. He outlined the arrangements he had made for collection and transportation.

Open forum 2

Staff grade

Dr P. V. Scott (*Redditch*) expressed concern about the intentions of the College, particularly the initial ruling that appointees in the Staff Grade must be in possession of the Fellowship. He felt that this was contrary to the spirit of the staff grade which was intended for the 'stuck doctor'. Now, he noted it was 'strongly recommended' that Staff Grade appointees possess Fellowship. His concern was that Staff Grade posts would turn out to be of a purely service nature and be without worthwhile elements to ensure job satisfaction. In discussion, Dr C. J. Barham (*East Grinstead*) expressed satisfaction that the College were taking a more flexible approach. Dr S. M. Lyons (*Belfast*) informed the meeting that the anaesthetics subcommittee of the Central Consultants and Specialists Committee were apprehensive about developments within the Staff Grade. The committee has been in contact with the College and expressed concern with work programmes and call rotas. He emphasised that these posts are not replacement registrars and asked for further information about problems with specific posts.

Dr P. Morris (*Manchester*) suggested that the main concern of the College of Anaesthetists was to ensure that appointees to the Staff Grade should have received an appropriate training, and that possession of a Fellowship was an indication of this. Dr W. R. MacRae supported this view, pointing out that appointees to the Staff Grade would hold post for up to 30 years and that they must show evidence of training and skills under these circumstances. No other specialty would make an appointment to a position where the practitioner would treat patients independently without the holder of the post being in possession of the appropriate higher qualification.

Dr Morris also stressed that Staff Grade would not normally be expected to do on-call work, yet hospital visits had revealed abuse of this proviso. He pointed out also that there is provision for study leave in the terms and conditions of service of the Staff Grade. He noted the role of the Regional Educational Adviser (REA) as a representative of the College. Differences in interpretation of the criteria for appointment may have been due to differences in the requirements. The first duty of the REA was to ensure that applicants are appointable and secondly to appoint the best candidate.

Dr Jane V. Freshwater (*Airdrie*) expressed concern that management may press for appointments to the Staff Grade on economic grounds. She also suggested that applicants in possession of the Fellowship could be appointed to Associate Specialist posts leaving the Staff Grade to fulfil manpower requirements. Dr D. J. Robinson (*St Mary's*) speaking as a member of his regional manpower committee expressed doubt about the College's attitude to appointments of candidates with and without Fellowship. An additional view was expressed that it did not matter whether Staff Grade anaesthetists worked at night as this made them feel a useful member of the department. In contrast, Dr S. M. Lyons (*Belfast*) felt that this was an uneconomic and wasteful way of using the talents of this

group. Dr Margaret L. Heath (*Lewisham*) felt that there should be a review of manpower strategies for the whole specialty.

NHS Trusts

Dr J. E. Charlton reviewed changes to the NHS Terms and Conditions of Service that had been proposed by NHS Trusts. The greatest concern had been expressed about attempts to 'gag' employees and stop the expression of personal opinions about the conduct of affairs within an NHS Trust. Dr P. M. Brodrick (*Mount Vernon*) confirmed the existence of these problems and informed the conference that he was not permitted to express his own views to the meeting; only those of the Mount Vernon NHS Trust. Dr J. A. T. Duncan (*Dunfermline*) outlined problems associated with the expression of the freedom of speech within medicine. He suggested strategies to cope with these problems.

There was considerable discussion about this matter and it was agreed that any attempt to limit fair and free comment about conditions and work practices in any part of the NHS was improper and should be resisted. Dr C. J. Bowley (*Nottingham*) asked that advice be available for consultants faced with these problems. Dr Rachel Hall (*Reading*) felt that this was a symptom of the conflict between the profession and management which would eventually lead to a progressive destruction of the strength of bodies such as the Association. She felt that Government should be lobbied to ensure that rights of individuals were protected. Dr D. Childs (*Chester*) cited evidence of a unilateral contract change by management and Honorary Secretary asked for full details.

Dr W. A. Lindsay (*Carshalton*) raised the idea of having block contracts for anaesthetic departments. The President confirmed that contracts of this nature had been agreed in some Health Districts and that they seemed to be a good idea as they permitted a more flexible approach to the work of the department. Dr A. G. Cole (*Leicester*) drew attention to split site and cross-Health Authority contracts. He felt that work of this sort was beneficial to individuals and departments, but anticipated that these current working practices would not be popular with managers. Dr P. J. Bickford-Smith (*Bradford*) expressed concern that the right of appeal to the Secretary of State would be removed in cases where there was a dispute between the clinician and the employing NHS Trusts. He feared that this could lead to suspension without pay. In addition, he outlined other possible scenarios whereby members might be disadvantaged by the introduction of local terms and conditions of service. Dr Bickford-Smith suggested that it might be prudent to create a database and hold details of variations in contract.

In closing the meeting the President expressed gratitude to the speakers for their valuable presentations. He thanked Linkmen for their many constructive comments and their continuing support of the Association and the vital links between the members and Council. The meeting was closed.

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EPIDURAL & SPINAL BLOCKADE IN OBSTETRICS

Edited by

Felicity Reynolds, Reader in Pharmacology Applied to Anaesthetics, Hon. Consultant, Anaesthetics, St. Thomas' Hospital, London, UK.

This book has evolved from the Obstetric Anaesthetist's Association meeting in the UK in September 1989. Unlike other books of proceedings however, the well-known contributors were invited not only to speak but also to provide a chapter reviewing their subject, prepared especially for the book.

Edited by Dr. Reynolds, the result is an interesting blend of a review of the last ten years' developments coupled with presentations of current research and coloured throughout with stimulating, amusing discussion.

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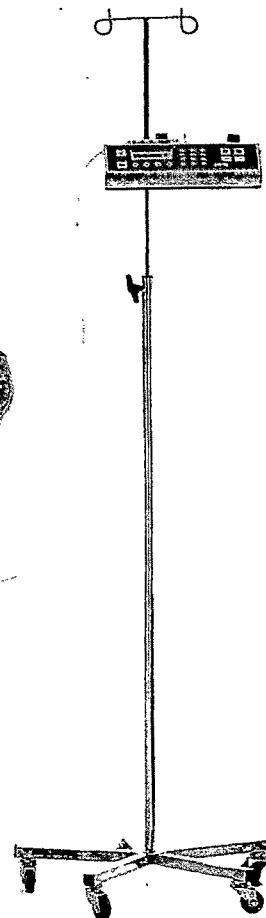


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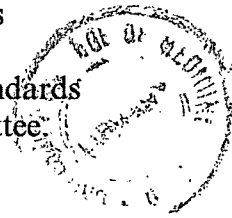
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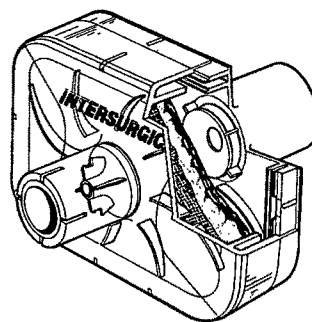
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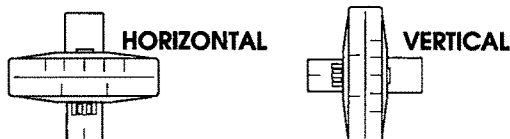
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Editorial

HIV and hepatitis—some way to go

In April 1988, the Association of Anaesthetists of Great Britain and Ireland issued a booklet entitled '*AIDS and hepatitis—guidelines for anaesthetists*' [1]. This was the product of a working group convened by the Association to consider all aspects of infection control in the context of anaesthetic practice. As most carriers of HIV and hepatitis are unidentified, the recommendations were intended to apply to the management of all patients. Since 1988, HIV infection has escalated in all countries and the number of known transmissions to health care workers, from their patients, has increased to more than 60 [2]. From prospective studies, the known risk of infection from percutaneous transfer of blood from a carrier of HIV remains constant (0.37%) [3]. A recent development, and a cause of much concern, was the description of transmission of HIV by a dental surgeon with AIDS to five of his patients apparently during the course of treatment [4-6]. On the hepatitis front, the safety and efficacy of recombinant hepatitis B vaccine continues to be apparent and it is anticipated that immunisation of staff engaged in invasive procedures will soon become mandatory. There is no vaccine for hepatitis C virus, which can also be transmitted by inoculation injury, and the prospects for an HIV vaccine seem remote.

It was considered important to reconvene the working group of the Association of Anaesthetists to review the current situation in the light of the spread of the AIDS epidemic and with further knowledge about the virus and its transmission. New guidelines have been produced and these will be issued soon. It is clearly important, although rarely possible, to assess the impact of published guidelines on work procedures. The two papers by O'Donnell and Asbury in this issue provide valuable, and alarming, insight into the perception of risks and the preventative measures adopted by a significantly large number of consultant and trainee anaesthetists in the United Kingdom. The important messages in the 1988 Guidelines document have either not reached the majority of members of the Association or have been seen but not adopted. As a virologist advising anaesthetist colleagues on the working group I have formed a false impression of what is happening generally within the Speciality. Most anaesthetists I know have altered their techniques in line with the 1988 Guidelines—clearly they are the converted.

It is sad to learn that anaesthetists are practising their own variety of 'Russian roulette' by exposing themselves to inoculation injury; the dangerous practice of resheathing needles is continued by more than one in three. The acceptance rate for hepatitis B vaccine is encouragingly high (87.2%); however many were unaware of the need to have their blood checked to ensure the vaccine has protected them (the non-responder rate is 5-10%) and I suspect they are not programmed to the need to have periodic booster doses.

Although apparently intact skin provides an effective barrier to HIV, hepatitis B virus can be highly infectious and there is the likelihood of infection via micro-cuts present in skin which may appear undamaged. The risk of transmission of hepatitis B virus from a high infecti-

vity (e antigen positive) carrier may be greater than 30% if a nonimmune health care worker is exposed by percutaneous inoculation [7-9]. The risks of percutaneous transmission of hepatitis C virus are unknown, although occupational infections of health care workers have been reported [10, 11]. It is therefore sensible to guard against the potential threat of transmission from unidentified carriers of these, and other, blood-borne viruses by wearing disposable gloves for all patients. Some of the arguments against attempting to screen all patients for HIV and hepatitis B virus are presented in the first paper by O'Donnell and Asbury. The General Medical Council has not sanctioned HIV testing without the fully informed consent of the patient [12]. Routine screening of patients (and health care workers) has not been supported by the Health Departments [13, 14] or the Royal College of Surgeons of England.

Some of the responses to the questionnaire were particularly disturbing. Only 17.7% of respondents took any form of personal history relating to infection risks (sexual habits, drug misuse) from patients pre-operatively. This seems anomalous in view of the expressed general level of concern about the threat of HIV and hepatitis, and particularly as the majority of anaesthetists seem to wish to continue an approach of spotting 'high risk' patients. It must come as a shock to many that approximately 20% of anaesthetists would be prepared to delay emergency surgery if there was a suspicion of HIV or hepatitis B infection.

What can be done to reduce the risks of occupational transmission of HIV and other blood-borne viruses to anaesthetists (and to patients)? The answer is surely to adopt Universal Precautions [15] which can be applied to all patients irrespective of the knowledge or suspicion of carrier status. The concept of Universal Precautions has been rejected, often without sufficient consideration, as being too cumbersome and economically non-viable, by a number of specialist groups. While they continue to pursue a policy of 'risk group' spotting in a two-tier approach to infection control, the numbers of infected individuals in the perceived 'low-risk' population is increasing.

The inconvenience and expense of adopting Universal Precautions are determined by the stringency and level of protection considered. It would be relatively non-intrusive and inexpensive for anaesthetists to introduce as a routine certain changes to their practice which would render them, and their patients, at a much lower risk of infection. The use of a fresh pair of disposable gloves for the induction of anaesthesia in each patient, and the further use of gloves for any subsequent invasive procedures, has been adopted by some anaesthetists and would not contribute excessively to the costs of the operation. The routine use of eye protection is being adopted increasingly by surgeons and dentists and would provide adequate protection of a potentially vulnerable portal of entry. Needles which have been in contact with the patient should not be resheathed and syringes with needles attached should not be handed directly to another person but placed into a tray for

transfer. All needles and other sharps should be placed in acceptably rigid disposal bins and not left lying around. Particular care should be taken to cover any cuts and abrasions with occlusive dressings and the danger of working with extensive skin lesions such as eczema or chapping should be fully realised. Blood from any patient should be regarded as potentially very hazardous and any spillage should be disinfected as soon as possible and the area cleaned, as laid down in local infection control policies.

The introduction of Universal Precautions is relevant to the protection of the patient as well as the anaesthetist. General adoption of simple, relatively inexpensive measures as outlined above is the best protection for the patient under the care of an anaesthetist who is a carrier of HIV or hepatitis B virus. The concept of regarding any patient as potentially infectious extends on to other areas of anaesthetic practice and particularly the need to ensure that infection is not transferred from one patient to another. Although there is no evidence for airborne spread of HIV and the hepatitis viruses, great care must be taken to ensure that blood transfer does not occur. Oropharyngeal airways, nasopharyngeal airways, tracheal tubes and contaminated breathing systems must either be disposed of, or sterilised adequately, between patients.

The reconvened working group at the Association of Anaesthetists were fortunate to have the data from the studies of O'Donnell and Asbury during the preparation of the revised Guidelines. The risks of occupational exposure to hepatitis B and HIV may be perceived to be low but the implications of acquiring these infections are profound. Apart from the immediate or delayed morbidity and mortality associated with them, development of a persistent infectious carrier state may have implications for continued employment. Occupational guidance issued by the UK Health Departments [14] recommends that HIV infected health care workers should not perform invasive techniques in which injury to the worker could result in the operator's blood contaminating the patient's tissues.

Studies of the acquisition of hepatitis B infection by patients from surgeons and dentists who are high infectivity carriers of the virus render it likely that immunisation will become mandatory and e antigen positive carriers working in surgery, dentistry and some other specialties will be required to restrict their practice or enter other branches of the profession. It is to be hoped that the new Guidelines document to be produced by the Association will have more impact than the last.

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Editorial notices

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The occupational hazard of human immunodeficiency virus and hepatitis B virus infection

I. Perceived risks and preventive measures adopted by anaesthetists: a postal survey

N. G. O'DONNELL AND A. J. ASBURY

Summary

We have conducted a postal survey of members of the Association of Anaesthetists to ascertain perceived risks and preventive measures adopted with regard to the occupational hazard of Human Immunodeficiency Virus and Hepatitis B Virus infection. Despite recognition of the infection risk and the adoption of appropriate measures when managing known infected patients, the majority of anaesthetists have not implemented simple precautions in their daily routine work. Less than 16% of respondents routinely wear gloves and more than one in three still resheath needles. It would appear that the recommendations of the Association with regard to universal safety precautions have not been implemented by the majority of its members.

Key words

Infection; human immunodeficiency virus, hepatitis B virus. Anaesthetists; risk, prevention.

In 1988, the Association of Anaesthetists published recommendations [1] which they felt should be introduced universally in anaesthetic practice to minimise the risks to anaesthetists from blood borne viruses, in particular Human Immunodeficiency Virus (HIV) and Hepatitis B Virus (HBV).

The incidence of HBV infection, and to a greater extent HIV infection, is increasing within the general population. By June 1991 there were almost 16 000 reports of HIV positive individuals in the UK [2]. The actual number of HIV positive individuals in the country is unknown, but is estimated to be between 36 000 and 148 000 [3]. This number is likely to increase in the future.

Currently, the risk of anaesthetic personnel contracting HIV from infected patients is small (the incidence of seroconversion following an inoculation injury involving an HIV positive patient is thought to be around 0.4% [4]). However, the risk will no doubt increase as the incidence of HIV positive individuals in the community increases. Inoculation with blood from HBV positive patients is much more likely to lead to infection; the reported incidence of seroconversion following an inoculation with blood from a HBV positive patient is approximately 5% [5]. In the case of hepatitis B 'e' antigen positive individuals, the incidence of seroconversion following an inoculation injury may be as high as 30% [6].

The majority of hospitals now have strict guidelines for the management of known HIV or HBV positive individuals presenting for anaesthesia and surgery. However, it is our impression that when dealing with the population at large, the majority of anaesthetists appear to adopt either no measures or inadequate measures to minimise any infection risk to themselves which may exist. There appears to be the assumption that all patients are HIV or HBV negative until proven otherwise.

We have conducted a postal survey of members of the Association of Anaesthetists to assess attitudes to infection and prevention. The specific objective of the survey was to determine the attitude of anaesthetists to the risk of HIV and HBV infection and to gauge their practices in the light of the Association's recommendations. It was hoped that the results of the survey would allow us to determine if the recommendations of the Association have been implemented by individual anaesthetists.

Methods

The questionnaire was initially tested on colleagues within the hospital. When reliable responses were obtained and recommendations with regard to equivocal questions incorporated, a final draft of the questionnaire was created. A total of 2525 questionnaires were distributed to consultant

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and trainee members of the Association of Anaesthetists, names having been chosen at random from the mailing lists. Members of the association residing in the Republic of Ireland or elsewhere overseas were not included in the study because of the difficulties posed by the postal services.

A prepaid return envelope was supplied with each questionnaire. This envelope was coded to allow follow-up of nonrespondents. Anyone who did not reply to the original questionnaire received a second, approximately 6–8 weeks after the original had been sent.

Data analysis

Information from questionnaires was checked, stored and analysed using Ashton Tate's dBase III+ package. The database was registered under the Data Protection Act.

All results are expressed as a percentage of the total number of respondents; in some tables and figures the total does not reach 100% because some respondents did not answer every question.

Statistical analysis

Results were analysed statistically using the Chi-squared test with Yates' correction as appropriate. Since we were specifically interested in any statistical difference in the frequency of affirmative answers, where appropriate, respondents answering 'no' or 'don't know' were grouped together. A probability of < 0.05 was considered to be statistically significant.

Results

One thousand nine hundred and ninety two of the 2525 questionnaires were returned and analysed, giving a response rate of 79%. Demographic data from the questionnaire is illustrated in Tables 1(a–c). Sixty seven percent of respondents were male and 27% female (6% did not state their sex).

Of those who responded to the questionnaire, 81% felt HIV posed some threat to them in their working environ-

Table 2. Anaesthetists in favour of routine pre-operative screening of patients for Human Immunodeficiency Virus (HIV) and Hepatitis B Virus (HBV).

Pre-operative screening		% of respondents
HIV	Yes	49.7
	No	41.9
	Don't know	7.8
HBV	Yes	51.8
	No	38.9
	Don't know	6.6

A greater number of respondents were in favour of pre-operative screening for HBV than HIV ($p < 0.05$).

ment; this decreased to 69% in the case of HBV. This difference in perceived risk was statistically significant ($p < 0.01$). More than 75% of respondents worked in hospitals where specific guidelines for the management of HIV or HBV positive patients existed (HIV 78%, HBV 79%).

Eighty seven percent of those responding to the questionnaire worked in hospitals where 'at risk' personnel were offered immunisation against HBV and 86% had been immunised. Although the numbers of those immunised is high, only 63% of this group were aware that their antibody titre was adequate to ensure protection. Eighty nine percent of all who replied stated that they would seek immunisation against HIV if an effective, safe vaccine was available. Significantly more individuals stated that they would seek immunisation against HIV, if available, than were immunised against HBV ($p < 0.01$).

Tables 2–4 illustrate the effect of the possible risk of HIV and HBV on pre-operative management. Approximately 50% of respondents (Table 2) were in favour of the routine screening of all patients pre-operatively, whenever possible, for both HIV and HBV. Around 70% of those who replied (Table 3) would cancel routine, nonurgent surgery until a result was available if there was some doubt about a patient's HIV or HBV status. In the case of emergency surgery this figure decreased, but was still approximately 20% (Table 4). With respect to pre-operative screening and delaying routine surgery, respondents were more concerned

Table 1(a–c). Demographic data.

(a)		(b)	
Age; years	% of respondents	Grade	% of respondents
20–29	11.9	Consultant	56.0
30–39	47.6	Senior Registrars	14.1
40–49	23.7	Registrar	19.1
50+	16.1	Senior House Officer	5.7
		Clinical Assistant	2.3
		Other	2.5
(c)			
Health Region	% of respondents	Health Region	% of respondents
Northern	4.8	West Midlands	7.7
Yorkshire	5.3	Mersey	4.8
Trent	8.3	North Western	6.6
East Anglia	3.5	Wales	4.9
Thames	23.1	Lothian	2.4
Wessex	3.9	Scotland (other)	9.6
Oxford	4.1	N. Ireland	3.9
South Western	5.1	M.O.D.*	1.6

*M.O.D. Employed by the Ministry of Defence.

Table 3. Anaesthetists who would delay routine surgery if there were doubt about a patient's Human Immunodeficiency Virus (HIV) or Hepatitis B Virus (HBV) status.

Delay		% of respondents
HIV	Yes	69.3
	No	26.0
	Don't know	4.2
HBV	Yes	71.8
	No	23.3
	Don't know	3.4

Respondents were more likely to delay surgery if there was concern about HBV status than HIV status ($p < 0.05$).

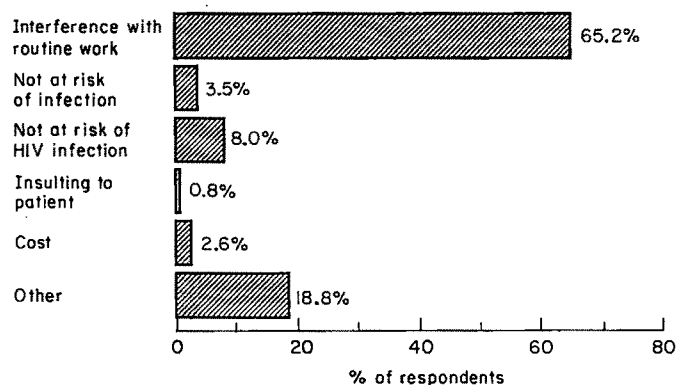
Table 4. Anaesthetists who would delay emergency surgery if there was doubt about a patient's Human Immunodeficiency Virus (HIV) or Hepatitis B Virus (HBV) status.

Delay		% of respondents
HIV	Yes	21.1
	No	75.3
	Don't know	3.3
HBV	Yes	20.9
	No	74.6
	Don't know	3.3

There was no significant difference between the number of respondents who would delay surgery if there was concern about a patient's HIV status as opposed to HBV status.

about HBV than HIV ($p < 0.05$). The difference with regard to delaying emergency surgery was not statistically significant. Only 18% of respondents took any form of personal history relating to infection risks (sexual habits, drug abuse) from patients pre-operatively.

Only 16% of those who replied wore gloves routinely during normal daily work. The reasons for routine glove wearing are shown in Figure 1, the commonest being to reduce the risk of infection to self (55%) and general hygiene (25%). Of the glove wearers, 56% were influenced in their decision to start wearing gloves by the appearance of HIV. However 40% of those who routinely wore gloves

**Fig. 2.** Principal reasons for not routinely wearing gloves expressed as a percentage of total number of respondents who do not routinely wear gloves.

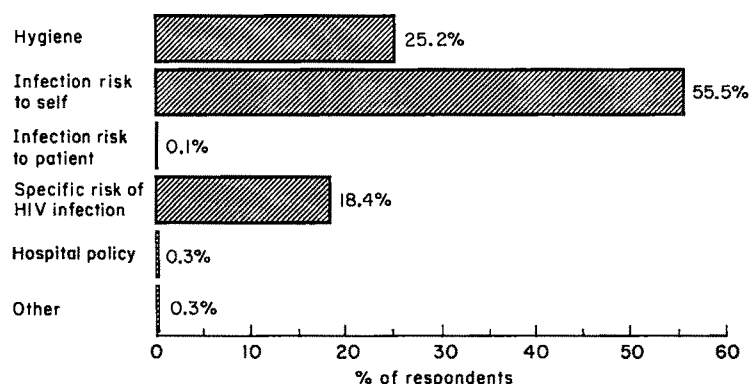
did not change their gloves between cases. Glove types routinely worn are shown in Table 5.

Reasons for not wearing gloves are displayed in Figure 2, by far the commonest (65%) being interference with routine tasks such as venous cannulation. Forgetfulness and lack of good quality, well-fitting gloves were the most commonly quoted 'other reasons' for not wearing gloves. Fifty eight percent of respondents not routinely wearing gloves stated they would do so if they thought it likely that their hands would be contaminated with the patient's blood.

Thirty five percent of respondents still resheathed needles which had been in contact with patient body fluids. Figure 3 demonstrates the incidence of needlestick injuries in a 6-month period immediately prior to completing the questionnaire. When glove wearers and nonglove wearers were compared, there was no significant difference in the inci-

Table 5. Glove type worn.

Type	% of respondents
Polypropylene	9.4
Nonsterile latex rubber	51.3
Sterile latex rubber	38.1

**Figure 1.** Principal reasons for routine glove wearing expressed as a percentage of the total number of respondents who routinely wear gloves.

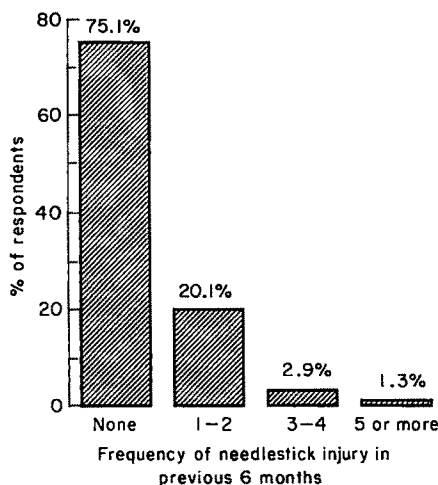


Fig. 3. Incidence of needlestick injuries sustained by individuals in the 6 month period immediately prior to completing the questionnaire. Values are expressed as a percentage of the total number of respondents.

dence of needlestick injuries between the two groups. Eighty five percent of individuals sustaining such injuries did not report the incident to occupational health or any

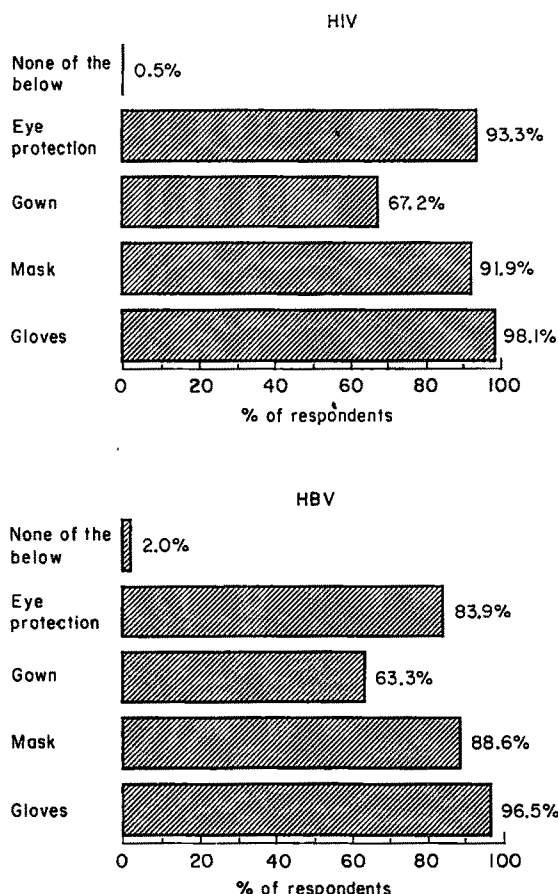


Fig. 4. Protective measures adopted by respondents when dealing with known Human Immunodeficiency Virus (HIV) or Hepatitis B Virus (HBV) positive patients. Values are expressed as a percentage of the total number of respondents. Respondents were more likely to wear gloves ($p < 0.01$), eye protection ($p < 0.01$) and gowns ($p < 0.05$) when managing known HIV positive patients. They were more likely to adopt none of the above measures when dealing with HBV positive patients ($p < 0.05$).

other appropriate authority. Of those who did not resheath needles, 59% were influenced in their decision by the appearance of HIV infection.

Figure 4 illustrates the protective measures adopted by respondents when anaesthetising a known HIV or HBV positive patient in circumstances in which aerosol contamination with blood from the operating site may occur. Respondents were more likely to wear gloves ($p < 0.01$), eye protection ($p < 0.01$), and gowns ($p < 0.05$) when managing patients who were HIV positive, and were more likely to adopt none of the above measures when managing HBV positive patients ($p < 0.01$).

Discussion

The results of our questionnaire suggest that individual anaesthetists feel at risk from blood borne virus infections; approximately 80% feel they are at risk from HIV and approximately 70% from HBV. This lower figure for HBV is repeated throughout our results and is statistically significant. We feel that this is a reflection of the high percentage of respondents who are immunised against HBV (86%). However, it must be remembered that protection following immunisation with the Hepatitis B vaccine provides only approximately 90% immunity to infection and then only once antibody titres are adequate. More than one in three of those immunised were unaware whether or not their titres were sufficient to provide protection. In addition, the 'risk groups' for carrying HBV are at a greater risk of being HIV positive than the general population. Therefore, even if antibody titres are satisfactory following immunisation against HBV, full precautions to prevent the risk of infection should be adopted when dealing with all patients. The incidence of immunisation against HBV in our study (86%) is higher than that reported in previous studies [7,8] and perhaps suggests that in recent years anaesthetists have become more aware of the value of immunisation.

Almost 50% of respondents were in favour of routine pre-operative testing of all patients for both HIV and HBV. This in itself would be a very costly undertaking and would create a number of major problems. For example, would the local laboratory services be able to cope with this number of requests for screening? Would we seek patient permission to undertake testing and, if yes, what would we do if they refused? Would we test them anyway or would we just refuse to anaesthetise them? Who would counsel those patients found to be HIV or HBV positive and how could we ensure patient confidentiality? Finally, in the case of HIV infection, there would be no way of ensuring that individuals were not being tested in the 'window period' of the infection when they were antibody negative but already infective.

Almost 70% of those who replied felt that they would delay routine, nonurgent surgery until a result became available, if there was doubt about a patient's HIV or HBV status and about 20% would delay emergency surgery. One could argue that this may be a reasonable course of action for routine, nonurgent surgery, but the problem is how to identify such patients? Less than 20% of anaesthetists take any personal history from patients relating to factors which may place them in 'high risk' groups and most patients are unlikely to volunteer the information. It would seem unreasonable to delay emergency surgery to await an HIV

or HBV result when, if sensible precautions are adopted, any risk to theatre personnel is minimal.

It would appear from our results that when dealing with known HIV or HBV positive patients (Fig. 4) the majority of anaesthetists adopt suitable precautions to minimise any infection risk, significantly more so in the case of HIV positive patients. Around 90% of respondents wear gloves and some form of eye protection in such circumstances. We believe the numbers in the case of HBV are lower for the reason already stated. Corneal inoculation with HBV has been described in laboratory animals [9], therefore the wearing of eye protection may be as important as gloves.

Although the majority of anaesthetists appear to adopt appropriate measures when dealing with known HIV or HBV positive individuals, our study suggests that the recommendations of the Association of Anaesthetists with regard to the universal adoption of safe practices have not been implemented by the majority of its members. Less than 16% of respondents wear gloves routinely for cannulation and intubation, although this is greater than the 8 to 9% reported in a recent smaller, local study [7]. By far the commonest reason for not wearing gloves was that they interfered with tasks such as cannulation. We feel, from personal experience, that with a little perseverance the minor handicap caused by glove wearing can be easily overcome.

Forgetfulness and poorly fitting gloves were also stated as reasons for failure to routinely wear gloves. However, this might be overcome by having dispensers of good quality gloves mounted on anaesthetic machines in the anaesthetic room and in theatre.

Almost 60% of those not routinely wearing gloves stated that they would do so if they thought it likely that contamination of their hands with blood might occur. It has been shown that this does indeed occur in a significant number of cases during routine anaesthetic practice [7].

Forty percent of glove wearers did not change gloves between cases. This is a practice that we feel should not continue. Repeated washing of gloves between cases breaks down the impervious barrier of the gloves. More importantly, the continued wearing of gloves will result in the contamination of case notes, theatre clothes and anaesthetic machines with any material present on the gloves. We would recommend that gloves are discarded before leaving the anaesthetic room and a new pair acquired, if necessary, for tasks undertaken in the operating theatre.

Despite recommendations from both the British Medical Association [10] and the Association of Anaesthetists [1] more than one in three of respondents still resheath needles which have been in contact with patient body fluids. Techniques for the 'safe' resheathing of needles have been described [11], but undoubtedly the safest means available is the immediate disposal of all sharps into an appropriate container. Needles must not be left uncapped in trays or on anaesthetic work surfaces because it has been shown that up to 40% of needlestick injuries may result from exposed needles [12]. This reinforces the argument for immediate disposal.

Some respondents said they did not wear gloves because they gave no protection against needlestick injury, and indeed suggested that the wearing of gloves may increase the incidence of needlestick injuries. We found no difference in the incidence of needlestick injuries between those who wear gloves and those who do not. The main reason

for glove wearing is to prevent contamination of any cuts and abrasions with blood. The best way to prevent needlestick injury is not to resheath needles but to dispose of them immediately into appropriate receptacles.

Almost 25% of respondents (Fig. 3) received at least one inoculation injury in the 6 months prior to the completion of the questionnaire. This compares with a 20% incidence during a 3 month period in a previous study [8]. Undoubtedly needlestick injuries are the major cause of transmission of blood borne viruses to health care workers. In addition, despite the legal requirement contained within the Health and Safety at Work Act (1974) to report such incidents to Occupational Health or another appropriate authority, almost 85% of anaesthetists fail to report any of these injuries.

In conclusion, it would appear that there is recognition of the existence of a risk with regard to blood borne virus infections among anaesthetists, resulting in the adoption of appropriate measures when anaesthetising known HIV or HBV positive patients. However, the majority of anaesthetists fail to use universal precautions during their routine daily work. Since it is now extremely difficult, if not impossible, to identify so called 'high risk' groups, we would recommend the wearing of gloves, and eye protection if a risk of splashing or aerosol formation exists, when anaesthetising all patients. In addition, we would recommend that gloves should be disposed of in the anaesthetic room and not worn throughout a case or used for more than one patient. The practice of resheathing needles should cease.

To date, it would appear that the recommendations of the Association of Anaesthetists have not been implemented by the majority of its members.

Acknowledgments

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The occupational hazard of human immunodeficiency virus and hepatitis B virus infection

II. Effect of grade, age, sex and region of employment on perceived risks and preventive measures adopted by anaesthetists

N. G. O'DONNELL AND A. J. ASBURY

Summary

The effect of grade, age, sex and region of employment on the attitude of anaesthetists to the possible risk of Human Immunodeficiency Virus (HIV) and Hepatitis B Virus (HBV) infection and the measures adopted to minimise the risk were assessed. As a group, anaesthetists in training were more concerned than consultants about the risk of HIV or HBV infection and, as a consequence, were more likely to adopt protective measures. A similar variation was seen with age, younger anaesthetists being more concerned about the risk of infection and adopting preventive measures in greater numbers than their older colleagues. The sex of the anaesthetist had minimal effect on their attitude. Despite the marked variation in the incidence of both HIV and HBV, the attitude of anaesthetists to the risk of infection and the numbers adopting simple preventive measures did not vary significantly on a regional basis throughout the country. However, there was a significant inter-regional variation in the availability and uptake of HBV immunisation ($p < 0.01$) and knowledge of the existence of local policy guidelines for the management of known HIV or HBV positive patients ($p < 0.01$).

Key words

Infection; human immunodeficiency virus, hepatitis B virus. Anaesthetists; risk, prevention.

In our previous paper [1] we presented the results of a survey of members of the Association of Anaesthetists undertaken to discover their attitude to the occupational risk of Human Immunodeficiency Virus (HIV) and Hepatitis B Virus (HBV). We also sought to discover what measures individual anaesthetists adopted in order to minimise any risk of infection which they felt existed. Our results suggest that, as a heterogeneous group, anaesthetists recognise the risk of occupational infection and the majority adopt appropriate measures when dealing with known or suspected HIV or HBV positive patients. However, the vast majority of anaesthetists have not implemented the recommendations of the Association of Anaesthetists [2] with regard to adopting universal precautionary measures when dealing with the general public at large. Fewer than 16% of anaesthetists responding to the survey routinely wore gloves and fewer than 18% attempted to discover if their patient was likely to be in an 'at risk' group for being HIV or HBV positive [1].

The incidence of HBV positive individuals in the UK is thought to be approximately 0.1–0.5% of the

population [2]. In the case of HIV, the total number of antibody positive individuals was recently reported to be just under 16 000 [3], but it has been estimated to be considerably higher (36 000 to 148 000) [4]. The incidence of HBV positive and, to a greater extent, HIV positive individuals varies greatly in different parts of the country (see below). The overall incidence of known HIV positive people in the country is approximately 1 in 3 500 (incidence of HIV positive individuals = 16 000; predicted UK population for 1991 = 57 000 000). The actual incidence may be considerably higher [4]. The incidence amongst women attending a London antenatal clinic has recently been shown to be as high as 1:230 [5] and in Edinburgh the incidence within the general population may be almost as high as this.

We have therefore analysed the responses of specific subgroups of anaesthetists who replied to the original questionnaire. The objective was to discover if there was a regional variation in attitudes towards possible infection risk and measures adopted to minimise any risk perceived to exist. In addition, we have analysed the results to see if

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grade, age or sex, like region of employment, affect the anaesthetist's attitude towards infection risk and the adoption of protective measures.

The distribution of respondents was as follows: 1331 were male, 540 were female and 121 did not state their sex. There were 1116 consultants and 774 trainees, of whom 281 were senior registrars, 380 were registrars and 113 were senior house officers.

Methods

Data analysis

The information already held on our database (Ashton Tate's dB3+) was further analysed to obtain specific data relating to how individual anaesthetists responded to each question depending on their grade, age, sex, and their region of employment. Since Edinburgh, at this time, is known to have a very high incidence of HIV positive individuals, Lothian was examined separately from the rest of Scotland.

Results were examined using the Chi-squared test, and a probability of < 0.05 was considered to be statistically significant.

As in our previous paper, we were specifically interested in any statistical difference in the frequency of affirmative answers, therefore, whenever appropriate, respondents answering 'no' or 'don't know' were considered as a single group for the purposes of analysis. Replies from respondents who had failed to state their grade, age, sex or region of employment were, where appropriate, excluded from analysis.

For ease of analysis of any variation in response by grade, respondents were classified as either consultants or trainees therefore replies from clinical assistants or others were not included in the analysis.

Data presentation

Values in tables and figures are expressed as a percentage of the total number of respondents. Cumulative values do not reach 100% because some respondents did not answer every question. Since the volume of data was extremely large we have only presented what we consider to be the important differences produced by the variables studied.

Results

Variation in response by grade

The data on effect of grade on perceived risks and the adoption of preventative measures are summarised in Table 1. A greater number of trainees wore gloves for routine work (19.4% compared to 13.1%, $p < 0.01$) but their decision to do so was less influenced by the appearance of HIV than consultants (46.0% as compared to 63.7%, $p < 0.01$).

There was no difference in the number of individuals who still resheathed needles in the two groups, but consultants were less likely than trainees to sustain needlestick injuries (incidence of needlestick injury 22.4% versus 27.3%, $p < 0.01$). There was no difference between the two groups as regards the reporting of needlestick injuries when they did occur.

The appearance of HIV infection was, for trainees, more of a factor in deciding to no longer resheath needles (63.1% compared to 53.3%, $p < 0.01$).

More trainees than consultants felt at risk from HBV (76.4% versus 64.0%, $p < 0.01$) and HIV (87.6% versus 76.2%, $p < 0.01$). Consequently, there was a higher incidence of immunisation against HBV among trainees (89.3% compared to 84.1%, $p < 0.01$) and trainees would be more likely to seek immunisation against HIV if a safe,

Table 1. Effect of grade on the attitude of anaesthetists to the risk of Human Immunodeficiency Virus (HIV) and Hepatitis B Virus (HBV) infection and the measures adopted to minimise such risk.

	Grade		p value
	Consultant	Trainee	
	% of respondents	% of respondents	
Routinely wear gloves	13.1	19.4	$p < 0.01$
Resheath needles	33.4	36.8	NS
Needlestick injury in previous 6 months	22.4	27.3	$p < 0.01$
Do not report needlestick injuries if they occur	85.2	83.9	NS
Immunised against HBV	84.1	89.3	$p < 0.01$
Would seek immunisation against HIV	86.3	93.0	$p < 0.01$
Feel at risk from HIV	76.2	87.6	$p < 0.01$
Feel at risk from HBV	64.0	76.4	$p < 0.01$
Ask personal history of patient re risk factors	14.9	21.7	$p < 0.01$
Pre-operative screen for HIV	54.8	41.9	$p < 0.01$
Pre-operative screen for HBV	55.4	46.1	$p < 0.01$
Delay routine, non-urgent surgery if patient ? HIV + ve*	72.1	64.9	$p < 0.01$
Delay routine, non-urgent surgery if patient ? HBV + ve†	72.8	70.3	NS
Delay emergency surgery if patient ? HIV + ve	23.8	17.1	$p < 0.01$
Delay emergency surgery if patient ? HBV + ve	23.4	17.2	$p < 0.01$

*HIV + ve = Human Immunodeficiency Virus positive;

†HBV + ve = Hepatitis B Virus positive.

Table 2. Effect of age on the attitude of anaesthetists to the risk of Human Immunodeficiency (HIV) and Hepatitis B Virus (HBV) infection and the measures adopted to minimise such risk.

	Age (years)				p value
	20-29	30-39	40-49	50+	
	% of respondents				
Routinely wear gloves	17.7	19.7	12.7	7.9	p < 0.01
Resheath needles	40.7	34.1	31.5	37.2	NS
Needlestick injury in previous 6 months	27.8	24.1	21.8	25.6	NS
Do not report needlestick injuries when they occur	89.4	81.6	86.4	88.1	NS
Immunised against HBV	94.1	88.5	85.4	74.4	p < 0.01
Would seek immunisation against HIV	97.0	91.2	87.5	83.3	p < 0.01
Feel at risk from HIV	89.9	84.3	78.1	70.7	p < 0.01
Feel at risk from HBV	75.8	74.5	68.6	64.9	p < 0.01
Ask personal history of patient re risk factors	25.7	17.1	15.2	17.1	p < 0.01
Pre-operative screen for HIV	40.3	44.3	54.1	67.7	p < 0.01
Pre-operative screen for HBV	44.9	49.0	56.3	68.3	p < 0.01
Delay routine, non-urgent surgery if patient ? HIV + ve*	65.3	65.4	75.1	77.0	p < 0.01
Delay routine, non-urgent surgery if patient ? HBV + ve†	70.6	69.9	77.6	75.7	p < 0.01
Delay emergency surgery if patient ? HIV + ve	15.7	17.9	22.0	35.0	p < 0.01
Delay emergency surgery if patient ? HBV + ve	15.3	18.4	21.6	32.2	p < 0.01

*HIV + ve = Human Immunodeficiency Virus positive;

†HBV + ve = Hepatitis B Virus positive.

reliable vaccine became available (89.3% versus 84.1%, $p < 0.01$).

A greater percentage of consultants than trainees were in favour of routine screening of patients for both HIV (54.8% versus 41.9%, $p < 0.01$) and HBV (55.4% versus 46.1%, $p < 0.01$). Consultants were also more likely to delay routine, non-urgent surgery until a result became available if there was concern about the patient's HIV status (72.1% compared to 64.9%, $p < 0.01$). There was no difference between the groups as to the number of individuals who would delay routine, non-urgent surgery if concern about the patient's HBV status existed.

In the case of emergency surgery, a greater number of consultants were in favour of delay if there was concern about either HIV (23.8% against 17.1%, $p < 0.01$) or HBV status (23.4% against 17.2%, $p < 0.01$).

Variation in response by age

The data relating to the effect of age on the perceived risk of HIV and HBV infection and preventive measures adopted are shown in Table 2. There was a significant difference ($p < 0.01$) in incidence of routine glove wearing in the different age groups, with the frequency of glove wearing being less among older anaesthetists. Age also affected the rate of HBV immunisation ($p < 0.01$) and the desire to seek immunisation for HIV if a vaccine became available ($p < 0.01$). The numbers in both cases decreased in the older age groups. The number of individuals who felt at risk from either HIV or HBV decreased as we moved up the age ranges. In the case of both viruses, the difference between the age ranges was significant ($p < 0.01$).

The desire for routine pre-operative screening for both HIV and HBV increased with increasing age, as did the wish to delay both routine, non-urgent and emergency

surgery, if doubt existed about the patient's HIV or HBV status. In each case, age had a significant effect on the attitude of the anaesthetist ($p < 0.01$).

Age did not affect the incidence of resheathing needles; the frequency of needlestick injuries or their reporting.

Variation in response by sex

The data on the effect of sex on the perceived risk of HIV and HBV infection and preventive measures adopted is summarised in Table 3. Females were more likely to obtain a relevant personal history from patients pertaining to the risk of infection (22.6% versus 15.9%, $p < 0.01$) and, as a group, felt more at risk from HBV than males (76.3% versus 67.0%, $p < 0.01$). Females more commonly stated interference with routine tasks as the principal reason for not routinely wearing gloves (70.5% against 63.0%, $p < 0.01$).

Male anaesthetists suffered fewer needlestick injuries during the 6 month period prior to completing the questionnaire (21.4% against 29.8%, $p < 0.01$).

The sex of the anaesthetist did not affect the frequency of, or reasons for, routine glove wearing; whether or not respondents resheathed needles; the rate of reporting of needlestick injuries; HBV immunisation or the desire to seek HIV immunisation if available; the risk posed by HIV; the wish for pre-operative screening for both HIV and HBV or the decision to delay routine, non-urgent or emergency surgery if there was concern about a patient's HIV or HBV status.

Variation in response by region of employment

Region of employment affected respondents answers to a number of questions and this is shown in Table 4. The

Table 3. Effect of sex of the anaesthetist on perceived risk of Human Immunodeficiency Virus (HIV) and Hepatitis B Virus (HBV) infection and the measures adopted to minimise such risk.

	Sex		p value
	Male	Female	
	% of respondents		
Routinely wear gloves	14.9	17.8	NS
Resheath needles	35.5	33.5	NS
Needlestick injury in previous 6 months	21.4	29.8	p < 0.01
Do not report needlestick injuries when they occur	84.9	83.9	NS
Immunised against HBV	86.7	85.9	NS
Would seek immunisation against HIV	88.7	90.7	NS
Feel at risk from HIV	80.8	81.7	NS
Feel at risk from HBV	67.0	76.3	p < 0.01
Ask personal history of patient re risk factors	15.9	22.6	p < 0.01
Pre-operative screen for HIV	49.9	48.7	NS
Pre-operative screen for HBV	50.4	55.2	NS
Delay routine, non-urgent surgery if patient ? HIV + ve*	70.6	66.5	NS
Delay routine, non-urgent surgery if patient ? HBV + ve†	72.4	71.5	NS
Delay emergency surgery if patient ? HIV + ve	21.5	20.2	NS
Delay emergency surgery if patient ? HBV + ve	20.9	20.9	NS

*HIV + ve = Human Immunodeficiency Virus positive;

†HBV + ve = Hepatitis B Virus positive.

percentage of anaesthetists in each region with knowledge of the existence of specific policy guidelines for the management of known HIV or HBV positive patients is shown in Figure 1. In both cases the variation between regions was statistically significant ($p < 0.01$). The knowledge among respondents of the availability of HBV immunisation for 'at risk' personnel and the rate of immunisation among anaesthetists also varied between regions ($p < 0.01$ in both cases; the data are shown in Fig. 2).

The likelihood of anaesthetists delaying emergency surgery if there was concern over a patient's HIV or HBV status also demonstrated an inter-regional variation ($p < 0.05$; the data is shown in Fig. 3).

Despite the large regional variation in the incidence of known HIV positive individuals (Table 5) there was only a borderline statistical difference in the number of respon-

dents from each region who felt at risk from HIV ($p = 0.053$). In addition there was no regional variation in the number of anaesthetists who felt at risk from HBV; were in favour of routine pre-operative screening for HIV or HBV; would wish to delay routine surgery if there was concern over HIV or HBV status; would seek HIV immunisation; routinely wore gloves or still resheathed needles.

Discussion

Our results suggest that consultants and trainees differ markedly in their attitude to HIV and HBV. Trainees view the possibility of occupational infection as a greater risk and are consequently more likely to be immunised against HBV and adopt universal preventive measures such as the routine wearing of gloves. Consultants, despite being less

Table 4. Effect of region of employment on the attitude of anaesthetists to the risk of Human Immunodeficiency Virus (HIV) and Hepatitis B Virus infection and the measures adopted to minimise such risk.

Effect	
Significant	Not significant
'At risk' personnel offered HBV immunisation ($p < 0.01$).	Incidence of routine glove wearing
Number of anaesthetists immunised against HBV ($p < 0.01$).	Reasons for not wearing gloves
Knowledge of adequate antibody titre following immunisation ($p < 0.01$).	Number of anaesthetists who resheath needles
Knowledge of management policy for known HIV or HBV + ve patients ($p < 0.01$).	Number of anaesthetists who would seek HIV immunisation
Failure to report needlestick injuries if they occur ($p < 0.05$).	Number of anaesthetists who feel at risk from either HIV or HBV
Number of anaesthetists who would delay emergency surgery if there was concern over the patient's HIV/HBV status, until a result was available ($p < 0.05$).	Number of anaesthetists in favour of pre-operative screening for HIV or HBV
	Number of anaesthetists who would delay routine non-urgent surgery until a result was available if there was concern about a patient's HIV/HBV status

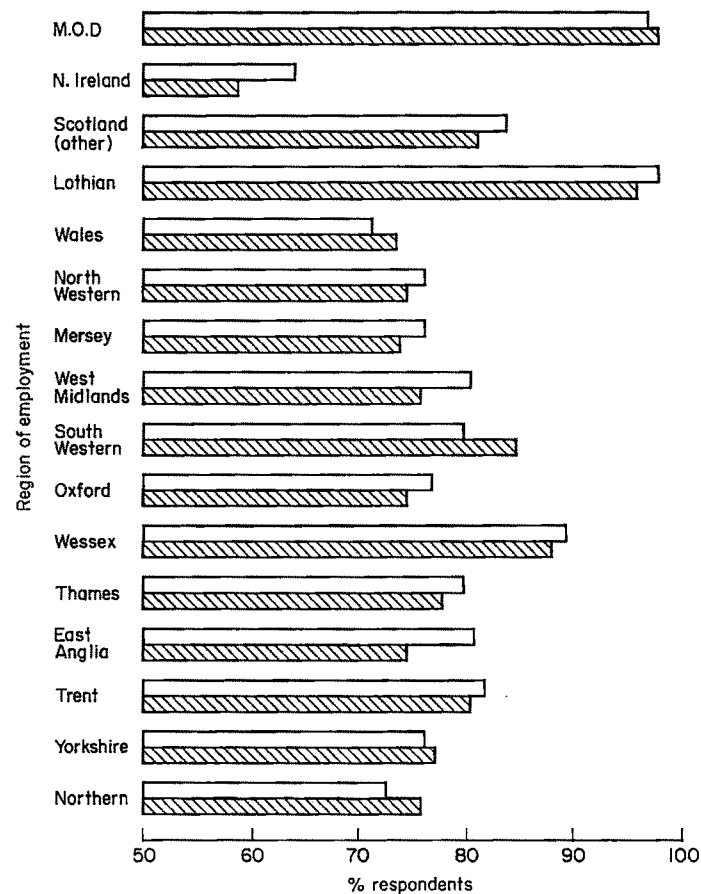


Fig. 1. Percentage of anaesthetists from each health region with knowledge of specific guidelines for the management of known Human Immunodeficiency Virus positive (striped bars) or Hepatitis B Virus positive (open bars) patients. In both cases there was a significant inter-regional variation ($p < 0.01$).

M.O.D. = employed by the Ministry of Defence.

Table 5. Incidence of known Human Immunodeficiency Virus (HIV) positive individuals in each health region and the percentage of anaesthetists, in each region, who felt at risk from HIV.

Region	Number of known HIV positive individuals (per 100 000 of population)	% of respondents who felt at risk from HIV*
Lothian	149.9	91.1
Thames	71.3	81.6
Scotland (other)	18.1	84.9
North Western	18.0	81.0
Oxford	16.9	74.4
Northern	12.0	85.7
Wessex	11.8	82.7
Yorkshire	11.7	84.2
South Western	10.6	81.6
West Midlands	10.4	79.1
East Anglia	10.1	82.1
Trent	9.0	81.0
Wales	8.3	81.9
Mersey	8.0	77.2
N. Ireland	5.4	66.7

Incidence of HIV-positive individuals calculated from the Public Health Laboratory Services AIDS Centre: Communicable Diseases Surveillance Centre and Communicable Diseases (Scotland) Units unpublished quarterly tables (No. 12, Sept. 1991) and the Office of Population Censuses and Surveys predicted populations for regional health authorities for 1991.

*Borderline significant difference between regions with regard to the number of respondents who felt at risk from HIV, $p = 0.053$.

NB No figures available for Ministry of Defence.

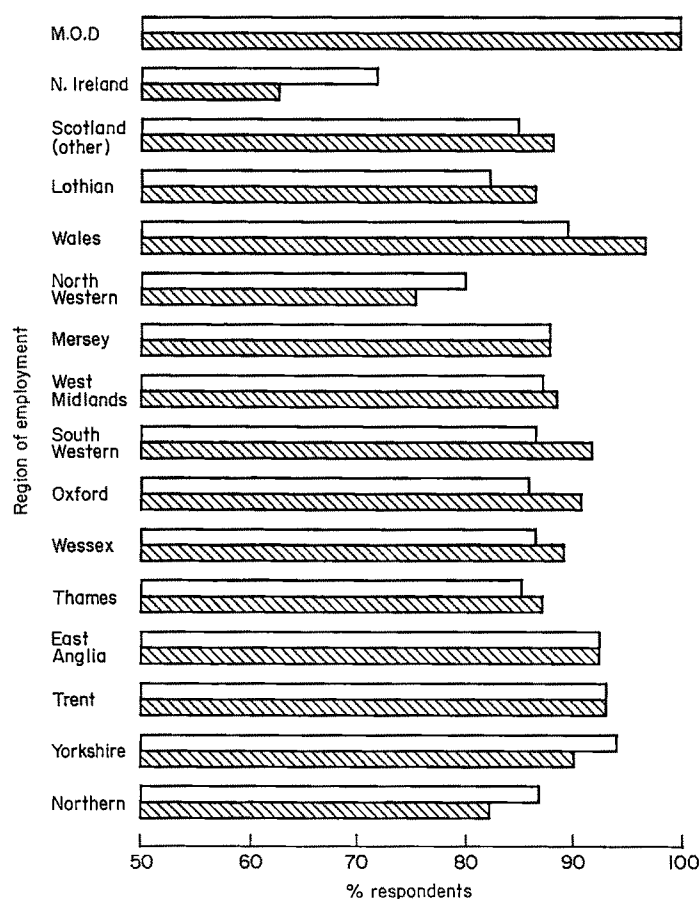


Fig. 2. Percentage of anaesthetists from each region with knowledge of the availability of Hepatitis B immunisation for 'at risk' personnel (striped bars) and the percentage of individual anaesthetists immunised against Hepatitis B (open bars). In both cases there was a significant inter-regional variation ($p < 0.01$). M.O.D. = employed by the Ministry of Defence.

concerned about possible risks from HIV or HBV, appear to be more conservative in their attitudes. They are more in favour of pre-operative screening for both HIV and HBV and more likely to delay even emergency surgery if concern exists about the patient's HIV or HBV status. They are, however, less likely to attempt to discover if their patient is likely to belong to an 'at risk' group. This difference in attitude we feel is unfortunate because many trainees will no doubt look to their senior peers for guidance in these matters and our results suggest that they will not necessarily receive advice along the lines of the Association's recommendations.

Interestingly, consultants were less likely than trainees to suffer needlestick injuries despite the fact that there was no difference between the groups with respect to numbers who resheathed needles.

Difference in age resulted in a picture similar to the trainee-consultant differences. Younger anaesthetists were more likely to feel at risk from HIV and HBV, routinely wear gloves and seek immunisation. These could all be a reflection of the acceptance that they may have to spend most of their career in anaesthetics with the spectre of occupational infection and its consequences. As the age of respondents increased they were less concerned about the risks from HIV or HBV but were also less willing to anaesthetise patient's whose HIV antibody or HBV antigen status was in doubt until a result became available.

There were few major differences between the sexes. Females were more concerned about the risks of HBV than males and also more likely to obtain personal histories from patients. Males were less likely to suffer needlestick injuries despite there being no difference in the incidence of resheathing needles. This may be due to the fact that a higher percentage of trainees, as compared to consultants, were female and, as already stated, trainees as a group were more likely to sustain needlestick injuries.

There was a definite regional variation with regard to knowledge of specific management plans for known HIV or HBV positive patients. Fewer respondents from areas where the incidence of HIV or HBV was likely to be low were aware of the existence of such policies (Fig. 1). This would suggest that either there are no firm local guidelines or, if they do exist, they are not well publicised amongst anaesthetists. Regions of the country where the incidence of HBV was lower than average were less likely to offer 'at risk' personnel immunisation, consequently fewer anaesthetists in these regions were immunised (Fig. 2). A regional variation with regard to delaying emergency surgery when concern about HIV or HBV status existed was also demonstrated, but this did not appear to relate to the local incidence of HIV or HBV (Fig. 3).

If individual regions are compared with each other then further differences can be demonstrated. Over the country as a whole, however, despite the marked variation in the

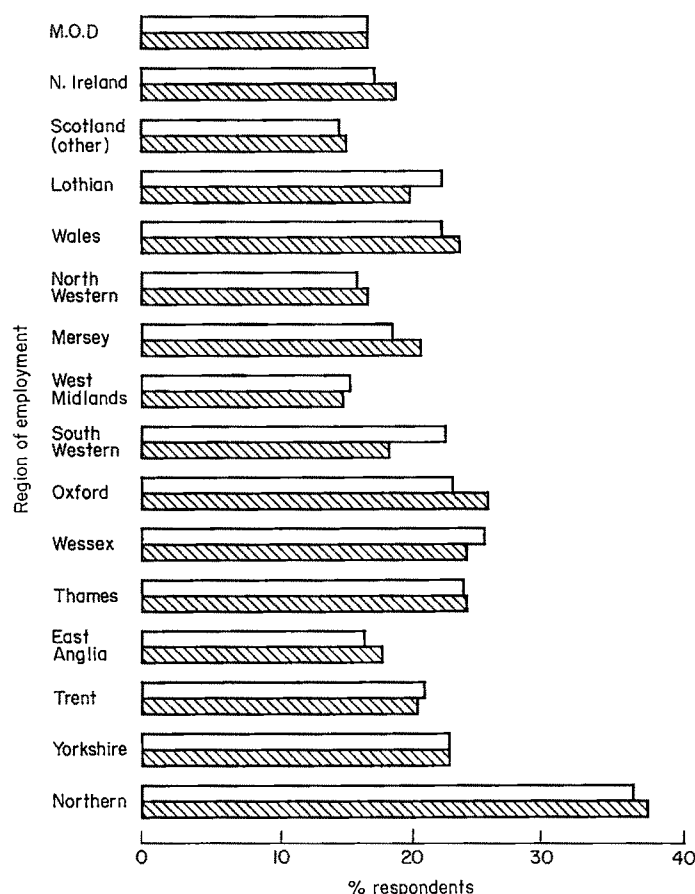


Fig. 3. Regional variation in the percentage of anaesthetists who would delay emergency surgery if doubt existed about a patient's Human Immunodeficiency Virus status (striped bars) or Hepatitis B Virus status (open bars). In both cases there was a significant inter-regional variation ($p < 0.05$).

M.O.D. = employed by the Ministry of Defence.

incidence of HIV and HBV positive individuals in different parts of the country, there was only a borderline statistical regional difference in the numbers who felt at risk from HIV, and no statistically significant difference in the numbers who felt at risk from HBV, the numbers in favour of pre-operative screening, the incidence of routine glove wearing or the number of individuals who still resheathed needles. This would suggest that the actual incidence of HIV or HBV positive individuals does not markedly affect the attitude of the individual anaesthetist with regard to infection risk or the adoption of preventive measures.

The variations in attitude which we have demonstrated with regard to grade, age and region of employment suggest the need to monitor progress in the future as the risk of blood-borne virus infection changes, both nationally

and regionally, and today's trainees become the consultants of tomorrow.

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Estimation of tidal volume from the reservoir bag

A laboratory study

P. R. KULKARNI, A. B. LUMB, M. W. PLATT AND J. F. NUNN

Summary

The accuracy of 21 anaesthetists in estimating tidal volumes from reservoir bag movements was assessed using a model lung apparatus. The breathing system configuration (Mapleson A or D), the grade of anaesthetist, and the years of anaesthetic experience had no effect on accuracy. Greater precision of tidal volume estimation was observed with larger tidal volumes and lower fresh gas flows. The mean systematic error of 18 of the 21 anaesthetists was greater than zero, indicating a general tendency to overestimate tidal volume. This study therefore strengthens the view that clinical observations should be supplemented with information from continuous monitoring devices.

Key words

Apparatus; reservoir bag.

Ventilation; tidal volume.

Anaesthetists spend a large proportion of their time monitoring spontaneously breathing patients using a combination of clinical observation of chest and reservoir bag movements and continuous monitoring devices [1]. However, anaesthetists often question the adequacy of tidal volume (V_T) estimation from simply watching the reservoir bag, and we are unaware of any study which indicates the precision of these observations. The size of reservoir bag excursion is not solely dependent on V_T , but is also likely to be affected by fresh gas flow rate and breathing system configuration. We therefore studied the expertise of anaesthetists of varying experience in assessing V_T under differing conditions of fresh gas flow and with two different breathing systems.

Methods

A ventilator (Medishield CPU 1) was set to drive the model lung described by Lyle *et al.* [2]. This was connected, in place of the patient, to either a Mapleson A (Magill) or Mapleson D coaxial (Bain) breathing system with a 2 l reservoir bag, so providing simulation of a spontaneously breathing patient. The ventilator was set to deliver tidal volumes of 100, 300, 500 and 700 ml with fixed respiratory frequency (breath.min⁻¹) and I:E ratio. The tidal volume delivered by the ventilator was validated with an 8 l wet

spirometer calibrated with a 1.5 l gas syringe, and shown to be within 10% of the desired V_T on five repeated measurements. The flow meters on the anaesthetic machine were set to give fresh gas flows (FGFs) of 3, 6, 9 and 12 l.min⁻¹.

Twenty-one anaesthetists of varying experience were recruited to the study and asked to estimate the tidal volume simply by observing a reservoir bag ('rebreathing bag'). The experiment was carried out during the course of a normal working day, provided the anaesthetist had not been on call the previous night. The aim of the experiment was explained to the subjects, who were asked to estimate V_T to the nearest 100 ml. They were also told that the maximum V_T was less than a litre. Each subject was seated in front of the anaesthetic machine with a screen placed between the anaesthetic machine and the model lung, such that the subject could visualise the fresh gas flow, the breathing system, and the reservoir bag, but not the ventilator or the model lung. Each anaesthetist estimated the V_T for the two circuits, the order of the two circuits being randomly allocated. Within each circuit the anaesthetist made 16 estimates, one of each combination of four tidal volumes and four FGFs, which were presented to the subject in random order.

The difference (in ml) between the estimated and actual V_T was considered the most appropriate variable for statistical analysis. The mean of these differences then represents

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the systematic error, whilst the SD of the differences is the random error. As a crude measure of whether the subjects' accuracy changed during the course of the experiment, the first and second systems presented to the anaesthetist (regardless of type) were compared. In order to do this, the total absolute difference (in ml) between the estimated and actual tidal volumes was calculated for each system and anaesthetist. For those anaesthetists tested on circuit A first, the difference between the second and the first period was calculated, while for those on circuit D first, the difference between the first and second periods was calculated. A Mann-Whitney test was performed comparing the group of anaesthetists with circuit A first with the group that had circuit D first.

The assumption of normally distributed residuals and equality of residual variances was tested using the Shapiro-Wilks test and the Schweders's test respectively. An analysis of variance (ANOVA) was then performed with the following factors; breathing system (Mapleson A or D), FGF (3, 6, 9 or 12 l.min⁻¹), tidal volume (100, 300, 500 or 700 ml), and anaesthetist grade (SHO, Registrar, Senior Registrar or Consultant).

Results

The differences between the estimated and actual V_T fulfilled the assumption of normally distributed residuals ($p = 0.87$) and equality of residual variances ($p = 0.15$). In the number of subjects studied there was no significant difference between the first and second circuits presented to the subjects ($p = 0.084$), or in the difference between breathing system A or D ($p = 0.598$). The ANOVA showed that there was a highly significant difference in the accuracy of estimation of V_T at different FGFs. With higher FGF, the systematic error increased, whilst the between-subject random error remained the same (Fig. 1). There was also a significant difference in the systematic error of estimation at different set tidal volumes, V_T being estimated most accurately at 700 ml and least accurately at 300 ml (Fig. 2). Again, random error in estimation of V_T was unchanged. For all tidal volumes and fresh gas flows studied, the mean systematic error for 18 of the subjects

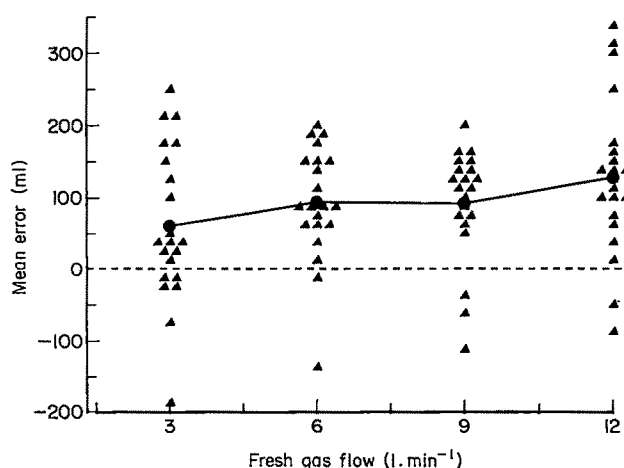


Fig. 1. Plot of error of each anaesthetist's estimated tidal volume against fresh gas flow (error = estimated minus actual tidal volume). ●, mean systematic error for all subjects; ▲, individual's systematic error.

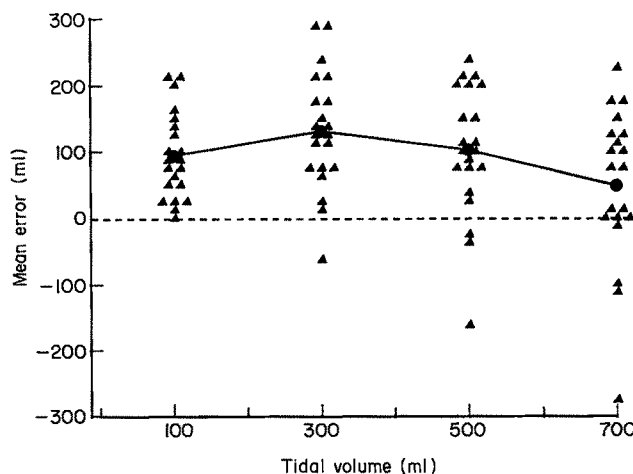


Fig. 2. Plot of error of each anaesthetist's estimated tidal volume against actual tidal volume (error = estimated minus actual tidal volume). ●, mean systematic error for all subjects; ▲, individual's systematic error.

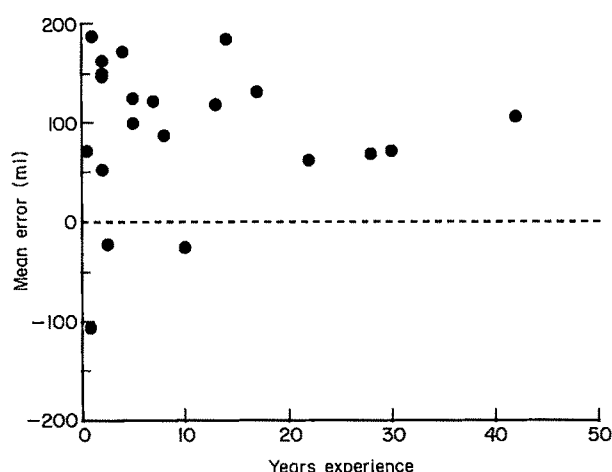


Fig. 3. Plot of years of experience of anaesthetist against each subject's overall mean systematic error of tidal volume estimation.

was positive. The grade of anaesthetist had no significant effect on accuracy of V_T estimation ($p = 0.981$), and from a plot of each subject's mean systematic error against years of experience, it would appear that this also has no effect (Fig. 3).

Discussion

The order in which the two breathing systems were assessed did not effect the accuracy of V_T estimation, indicating that there was no fatigue or learning curve as the experiment proceeded. Our clinical impression, that estimation may be less accurate with the Bain system, was not supported by this study. We can partly explain this by the fact that, in the clinical setting, anaesthetists need to use a much higher FGF for spontaneously breathing patients with the Bain breathing system than with the Magill system to prevent rebreathing. The predictable finding, that FGF affected the accuracy of V_T estimation, can be explained by the reduced excursions of the reservoir bag with increasing FGF. This finding agrees with the effect of V_T accuracy improving with larger tidal volumes which result in increased bag excursions.

Contrary to our expectations, grade of anaesthetist or duration of anaesthetic experience had no significant effect on the error of estimation, although from Figure 3 it appears that anaesthetists with more than 20 years experience have smaller mean differences. In this study, regardless of the actual V_T and FGF, the anaesthetists generally overestimated V_T , with only three out of 21 subjects having a mean systematic error of less than zero (Fig. 3). We can only speculate as to why there is such widespread overestimation of V_T , but this finding does seem to indicate that anaesthetists are optimistic regarding the adequacy of their patients' ventilation. Previous studies, attempting to assess the ability of anaesthetists to monitor clinically the respiratory system, have focused on detecting changes in pulmonary compliance, from palpation of the reservoir bag, the so-called 'educated hand' [3–5]. These studies, of both adult [3,4] and paediatric [5] anaesthetic practice, have shown the 'educated hand' to be a myth, and that even very experienced anaesthetists cannot estimate the patient's lung compliance. Our study shows that the ability of the 'educated eye' of most anaesthetists to estimate the

V_T visually is also a myth. It therefore strengthens the view expressed recently that clinical observations should be supplemented with information from continuous monitoring devices [1,6].

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Anaesthesia for myocardial revascularisation

A comparison of fentanyl/propofol with fentanyl/enflurane

S. M. UNDERWOOD, S. W. DAVIES, R. O. FENECK AND R. K. WALESBY

Summary

We studied the effects on myocardial performance and metabolism of fentanyl/propofol and fentanyl/enflurane anaesthesia in 20 patients before coronary artery bypass grafting. Anaesthesia was induced with fentanyl $20 \mu\text{g.kg}^{-1}$ and pancuronium 0.15 mg.kg^{-1} . Patients received, by random allocation, either propofol by infusion, $6 \text{ mg.kg}^{-1}.\text{h}^{-1}$ reduced by half after 10 min then adjusted as necessary (mean rate $2.8 \text{ mg.kg}^{-1}.\text{h}^{-1}$), or enflurane 0.8% inspired concentration for 10 min reduced to 0.6% and adjusted as required (mean 0.7%). Measurements were made before induction, after tracheal intubation, after skin incision and after sternotomy. There were no significant differences between the groups in any haemodynamic variables during the study. Following intubation both groups showed a rise in heart rate ($p < 0.01$) and cardiac index ($p < 0.05$). Systemic vascular resistance decreased after intubation ($p < 0.05$) then returned to baseline during surgery; stroke index was unchanged after intubation but was reduced during surgery ($p < 0.01$) as systemic vascular resistance increased. Regional and global coronary blood flow were maintained in both groups, as were myocardial oxygen consumption and lactate extraction ratio. However, lactate production did occur in one patient receiving enflurane and Holter monitoring confirmed ischaemia. One patient receiving propofol showed lactate production not accompanied by any ECG changes. This study suggests that propofol may be a suitable alternative to enflurane as an adjunct to opioids in anaesthesia for coronary artery bypass grafting.

Key words

Anaesthetics, intravenous; propofol.
Anaesthetics, volatile; enflurane.

Patients presenting for coronary artery bypass grafting are at risk of myocardial ischaemia and peri-operative infarction [1]. An anaesthetic technique which minimises these risks is clearly desirable. Haemodynamic changes which adversely affect myocardial oxygen balance or precipitate an adverse redistribution of coronary blood flow leading to 'intracoronary steal' are, therefore, to be avoided whenever possible.

Of the anaesthetic agents in common use halothane [2], enflurane [3], fentanyl and its derivatives [4-6], nitrous oxide [7] and isoflurane [8] have all at some time been associated with either haemodynamic effects likely to promote myocardial oxygen imbalance, experimental evidence of ventricular wall motion abnormality, or direct metabolic or electrophysiological evidence of anaerobic cardiac metabolism.

The haemodynamic effects of propofol have been described previously in patients with ischaemic heart disease following both intravenous bolus [9] and continuous infusion administration [10]. In this study we

compare the effects of fentanyl and a continuous infusion of propofol, with fentanyl and enflurane. We have restricted our study to the period between premedication and the start of cardiopulmonary bypass; we have examined the effects of anaesthesia and surgery on cardiac metabolism and systemic and coronary haemodynamics in both groups of patients.

Methods

Twenty patients who were scheduled for elective coronary artery bypass graft surgery and who gave written informed consent were studied with the approval of the ethics committee of the National Heart and Chest Hospitals. Patients were eligible if they were not over 70 years of age and had a left ventricular ejection fraction of greater than 30% as assessed by cineangiography. Patients were not studied if they had severe respiratory, hepatic, renal, haemopoietic or endocrine dysfunction, left coronary main stem stenosis or ventricular aneurysm, allergy to the

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study drugs or their pharmaceutical constituents, pregnancy or gross obesity.

All patients continued their anti-anginal medication up to and including the day of surgery and received anaesthetic premedication of diazepam 10–20 mg orally 3 h pre-operatively, followed by papaveretum 10–20 mg with hyoscine 0.2–0.4 mg intramuscularly 2 h before induction of anaesthesia. (The investigation took place before the current restrictions on the use of noscapine containing medications were introduced by the Medicines Control Agency.)

All patients had a 7 F balloon tipped thermodilution pulmonary artery catheter (Ecosse Ltd) and a 7 F thermodilution coronary sinus catheter (Electrocatheter Corporation, New Jersey) introduced into the right internal jugular vein. Both catheters were placed under X ray control and the position of the coronary sinus catheter was confirmed both by injection of radiopaque contrast and by measurement of oxygen saturation in blood withdrawn from it. Both catheters were placed under local anaesthesia and, when necessary, up to 2 mg midazolam were given intravenously during the procedure.

A radial arterial catheter was inserted, standard lead II of the electrocardiograph (ECG) displayed and continuous recording of lead V₃ started on a Holter monitor.

Measurements were made: (1) immediately before induction of anaesthesia; (2) after tracheal intubation; (3) after skin incision; (4) after sternotomy. The measurements listed in Table 1(A) were made at all the above stages and those in Table 1(B) were made at times 1, 2 and 4 only.

All vascular pressures were displayed using Spectramed P50 transducers and either Hewlett-Packard or Hellige bedside monitors. Cardiac output was calculated using bolus thermodilution injection in accordance with recommended practice. Multiple measurements were made at end-expiration and the mean of the series was recorded. Coronary sinus and great cardiac vein blood flows were calculated by the continuous thermodilution technique described elsewhere [11, 12]. The thermodilution curves were displayed on a three-channel printer and examined for evidence of artifact or instability. Measurements were made in duplicate and the mean recorded.

At each measurement point for metabolic parameters, blood samples were taken from the coronary sinus, great cardiac vein and radial artery. Haemoglobin concentration and oxygen saturation were measured with an oximeter (Radiometer OSM 2 Hemoximeter), blood gases with an ABL3 blood gas analyser (Radiometer, Copenhagen) and lactate concentrations using an enzymatic UV-system (Boehringer Mannheim GmbH).

Table 1. Haemodynamic and metabolic measurements.

(A) Haemodynamic
Heart rate
Systemic blood pressure
Right atrial pressure
Pulmonary artery pressure
Pulmonary capillary wedge pressure
Cardiac output
(B) Metabolic
Coronary sinus blood flow
Great cardiac vein blood flow
Arterial, coronary sinus, great cardiac vein
blood for: Hb, O ₂ saturation, P _a O ₂ lactate concentration

Anaesthesia was induced with fentanyl 20 µg.kg⁻¹ and 0.15 mg.kg⁻¹ pancuronium was given to provide neuromuscular blockade. The larynx was sprayed with 4% lignocaine 1 mg.kg⁻¹ before tracheal intubation and the lungs were ventilated with 50% oxygen in nitrogen throughout.

Patients were randomised to receive either propofol or enflurane anaesthesia after the postintubation measurements were completed. Propofol was given as an undiluted intravenous infusion starting at a rate of 6 mg.kg⁻¹.h⁻¹ for 10 min, reduced to 3 mg.kg⁻¹.h⁻¹ thereafter. Enflurane was commenced at an inspired concentration of 0.8% for 10 min and was then reduced to 0.6%. Both the propofol and enflurane regimens could be adjusted if clinically required.

Systemic blood pressure was maintained between 80 and 100% of the baseline value established before induction of anaesthesia. Perioperative systemic hypertension was treated when necessary with intravenous nitroglycerine in a bolus of 0.125 mg.

Coronary vascular resistance and myocardial metabolic indices were calculated using the formulae in the Appendix. Statistical analysis was performed using Freidmann two-way analysis of variance by ranks, Wilcoxon matched pairs signed-ranks and Mann-Whitney tests as appropriate.

Results

Patient data are shown in Table 2. The groups were comparable with respect to age, body weight, height and pre-operative anti-anginal medication, which consisted of combinations of nitrates, β -blockers and calcium channel antagonists. All the patients were men except for one woman in the enflurane group. The mean infusion rate of propofol, after the initial 10 min, was 2.8 mg.kg⁻¹.h⁻¹ and the mean inspired concentration of enflurane was 0.7%.

Changes in systemic blood pressure and vascular resistance are shown in Figure 1. There was no significant change in blood pressure following intubation, although there was a reduction in vascular resistance ($p < 0.05$) at that time. Blood pressure decreased in both groups after skin incision, a change more marked in the propofol group ($p < 0.001$). Five patients receiving enflurane and three administered propofol required bolus doses of glyceryl trinitrate shortly after commencement of surgery. Systemic vascular resistance rose above baseline in both the propofol and enflurane groups after sternotomy.

Figure 2 shows measures of right heart function. Right atrial pressure remained unchanged in both groups. The administration of either propofol or enflurane was associated with a reduction in pulmonary artery pressure ($p < 0.05$). Pulmonary vascular resistance rose after induction and intubation in the propofol group, but this was before starting propofol. Pulmonary vascular resistance decreased in both groups on commencement of the anaesthetic maintenance agent, as did right ventricular stroke work index.

Table 2. Physical characteristics (mean, SD) for propofol ($n = 10$) and enflurane ($n = 10$) groups.

	Age (years)	Weight (kg)	Height (cm)
Propofol	59 (7.5)	71 (12.6)	168 (7.5)
Enflurane	59 (7.2)	80 (18.0)	175 (11.8)

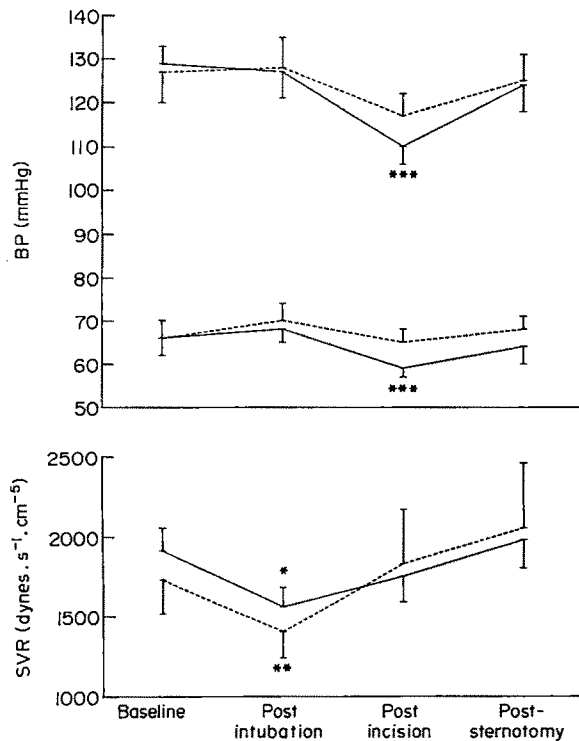


Fig. 1. Changes in mean (SE) values of systemic blood pressure (BP) and systemic vascular resistance (SVR) in the enflurane (---) and propofol (—) groups. * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$.

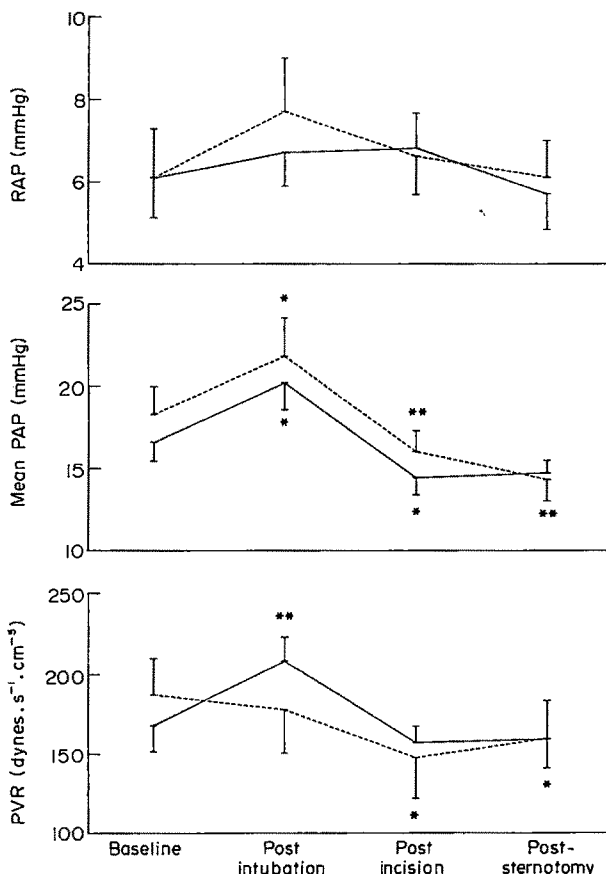


Fig. 2. Changes in mean (SE) values of right atrial pressure (RAP), pulmonary artery pressure (PAP) and pulmonary vascular resistance (PVR) in the enflurane (---) and propofol (—) groups. * = $p < 0.05$, ** = $p < 0.01$.

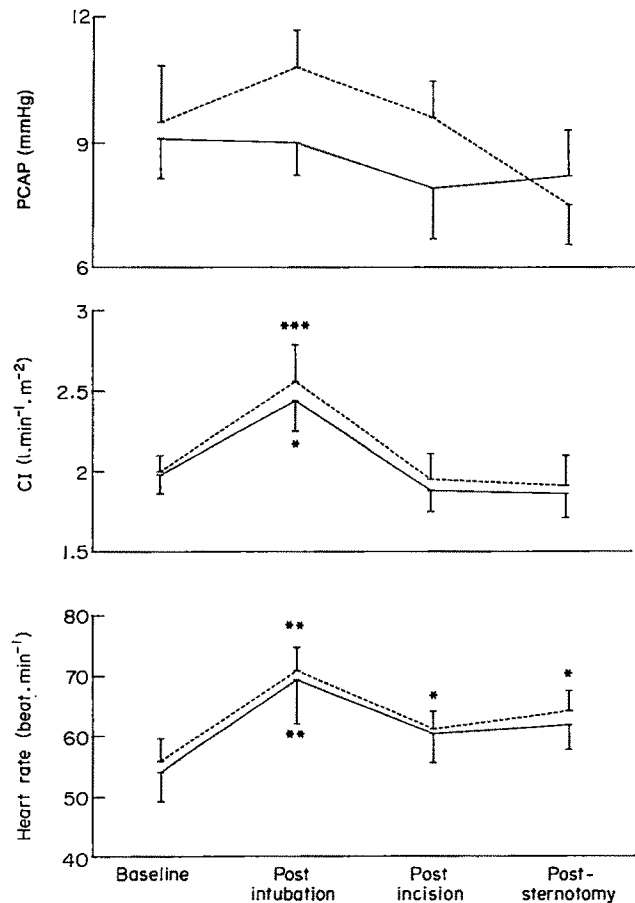


Fig. 3. Changes in mean (SE) values of pulmonary artery wedge pressure (PCWP), cardiac index (CI) and heart rate (HR) in the enflurane (---) and propofol (—) groups. * = $p < 0.05$, ** = $p < 0.01$, *** = $p < 0.001$.

Heart rate, pulmonary artery wedge pressure and cardiac index (CI) are shown in Figure 3. Cardiac index was increased after induction and intubation due to an increase in heart rate ($p < 0.01$) with little change in stroke volume index. However, after skin incision and sternotomy, during propofol or enflurane anaesthesia, cardiac index decreased as stroke volume index was reduced while the heart rate remained above baseline. Pulmonary artery wedge pressure was not significantly changed in either group although it was more stable with propofol.

In both groups these changes were accompanied by a reduction in left ventricular stroke work index ($p < 0.05$). Mean myocardial oxygen consumption and lactate extraction were unaltered (Fig. 4).

Coronary sinus blood flow increased after the introduction of enflurane, great cardiac vein blood flows remained above baseline and coronary vascular resistance was reduced (Fig. 5), but none of these changes reached statistical significance.

One patient showed lactate production after sternotomy, while receiving enflurane (Table 3). Coronary sinus blood flow was increased, as was myocardial oxygen consumption when lactate production was evident. Systemic vascular resistance had increased from an already high baseline level and cardiac index was reduced. ST segment elevation of 0.5 mm occurred at the same time. Another patient in the enflurane group exhibited lactate production in a sample from the great cardiac vein following intubation, before

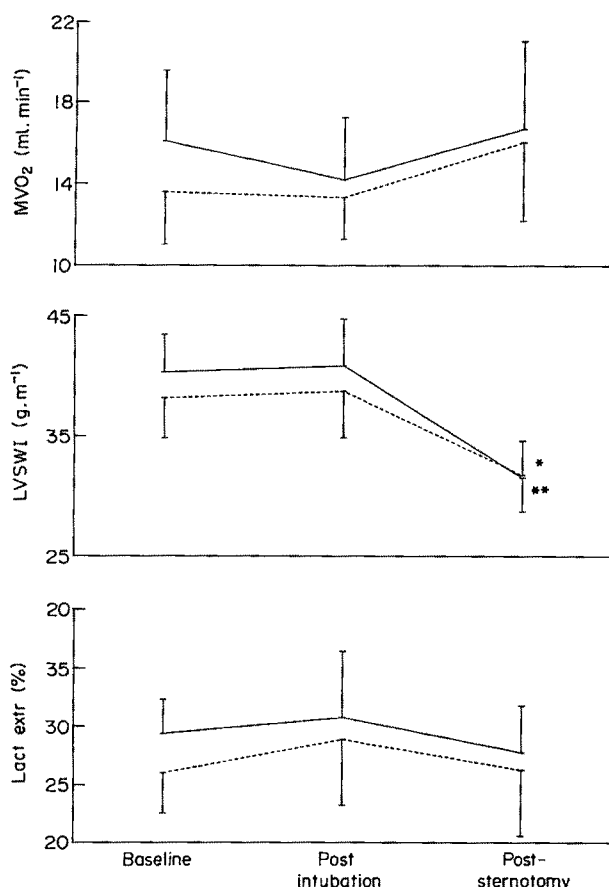


Fig. 4. Changes in mean (SE) values of myocardial oxygen consumption (MVO_2), left ventricular stroke work index (LVSWI) and myocardial lactate extraction (Lact extr) in the enflurane (---) and propofol (—) groups. * = $p < 0.05$, ** = $p < 0.01$.

enflurane administration, which improved after commencement of enflurane. At the time of lactate production, myocardial oxygen consumption and coronary sinus blood flows were above baseline. ST segment elevation of 0.7 mm occurred in the postintubation period but then resolved.

One patient in the propofol group became a lactate producer in the post-sternotomy period, as measured in

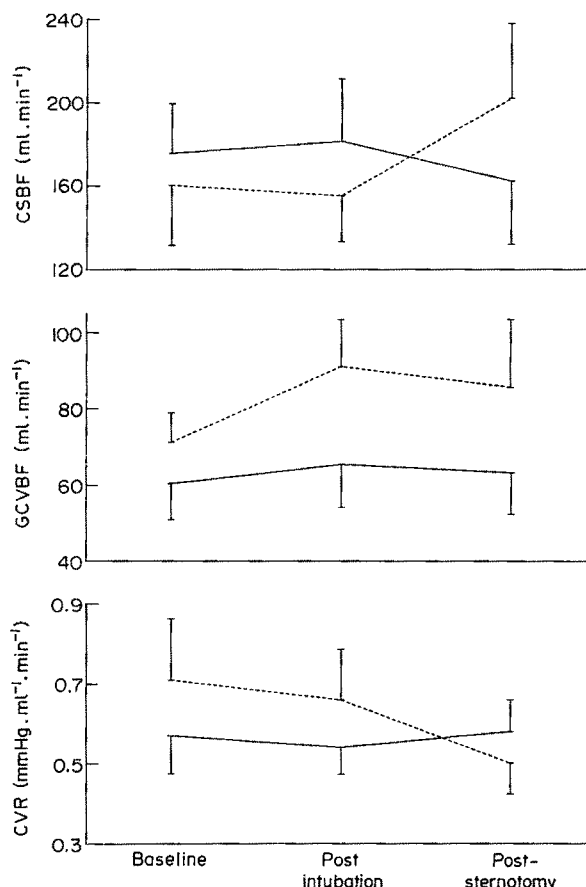


Fig. 5. Changes in mean (SE) values of coronary sinus and great cardiac vein blood flow (CSBF and GCVBF) and coronary vascular resistance (CVR) in the enflurane (---) and propofol (—) groups.

blood from the great cardiac vein. Great cardiac vein blood flow was increased as was myocardial oxygen consumption when lactate production occurred in that region. There were no ECG changes.

No adverse effects on myocardial conduction were seen during the study. PR, QRS and QTc intervals, shown in Table 4, remained within normal limits in both groups of patients.

Table 3. Data for one patient in the enflurane group.

	SVR (dynes.s.cm ⁻⁵)	CI (l.min ⁻¹ .m ⁻²)	MVO_2 (ml.min ⁻¹)	CSBF (ml.min ⁻¹)	MLE %
Baseline	3478	1.56	9.4	94	15
Postintubation	2735	1.87	11.3	103	39
Post-sternotomy	5452	0.92	13.0	166	-8

SVR, systemic vascular resistance; CI, cardiac index; MVO_2 , myocardial oxygen consumption; CSBF, coronary sinus blood flow; MLE, myocardial lactate extraction.

Table 4. Mean (SD) myocardial conduction times for propofol (P) and enflurane (E) groups.

Interval (s)	Group	Baseline	Postintubation	Postincision	Post-sternotomy
PR	P	0.19 (0.03)	0.17 (0.02)	0.19 (0.03)	0.19 (0.03)
	E	0.17 (0.03)	0.16 (0.03)	0.16 (0.03)	0.18 (0.02)
QRS	P	0.07 (0.01)	0.07 (0.02)	0.07 (0.02)	0.07 (0.02)
	E	0.07 (0.01)	0.07 (0.01)	0.07 (0.01)	0.07 (0.01)
QTc	P	0.39 (0.03)	0.40 (0.03)	0.40 (0.04)	0.39 (0.03)
	E	0.41 (0.05)	0.42 (0.04)	0.39 (0.02)	0.41 (0.03)

Discussion

There has been much interest recently in the influence of anaesthetic agents on the development or prevention of acute intra-operative ischaemia in patients with proven significant coronary artery disease. Previous work has suggested that close attention to those factors known to affect myocardial oxygen balance may minimise the risk of ischaemia [13] and early work led to the controversial conclusion that a degree of myocardial depression during anaesthesia was of benefit, particularly to coronary artery bypass graft patients with well preserved left ventricular function, since this would reduce myocardial oxygen demand and help prevent ischaemia [14,15]. However, since then the relative merits of different anaesthetic techniques have become more difficult to assess. Work on high dose opioid anaesthesia has shown that it can avoid haemodynamic depression in patients undergoing coronary artery bypass grafting [16,17], but there is also conflicting evidence that unacceptable ischaemic episodes occur [18,4].

Experimental and clinical studies with different volatile agents have also proved inconclusive. Data from studies with halothane have suggested that it may be a useful volatile supplement for patients with ischaemic heart disease, although this is controversial [2,19]. The use of isoflurane has provoked considerable controversy. Despite its systemic vasodilator effects, isoflurane anaesthesia has been associated with a significant incidence of ischaemia which has been attributed to its ability to dilate small coronary vessels, thereby inducing intracoronary steal in certain patients predisposed by their coronary anatomy [8,20]. However, there is also evidence that isoflurane-related ischaemia may result from either hypotension or reflex tachycardia as a result of its potent vasodilator effect [21]. Enflurane does not appear to possess as potent negative inotropic or dromotropic effects as those of halothane [22], nor does it cause systemic or coronary vasodilatation to the same degree as isoflurane [23,20]. A balanced anaesthetic technique using fentanyl and enflurane, similar to that described in this study, has been recommended for patients undergoing coronary artery bypass graft surgery [24]. We have used this technique as the 'gold standard' with which to compare maintenance of anaesthesia with propofol.

Our study was restricted to the period from induction of anaesthesia to commencement of cardiopulmonary bypass. It is clear from other work that peri-operative infarction is more commonly associated with adverse surgical factors than haemodynamic or other anaesthetic factors [25]. Ischaemic changes seen during bypass and after myocardial revascularisation are most likely to be due to surgical factors and render comparative assessment of anaesthetic agents at these times very difficult to interpret. Recent data have suggested that pre-bypass ischaemia is significantly more important than post-bypass ischaemia as a determinant of peri-operative infarction [26], but these data were obtained using highly sophisticated imaging techniques and their clinical relevance is not clear [27]. Nonetheless it would appear that during the pre-bypass period, when surgical factors are not implicated, the patient is at serious risk from ischaemia precipitating eventual infarction; thus this period represents the ideal time to study the comparative effects of anaesthetic techniques in patients with ischaemic heart disease.

The hemodynamic changes seen in this study were not marked and there were few differences between the two groups. Following induction of anaesthesia and tracheal intubation, but before either propofol or enflurane had been given, there was a significant increase in cardiac index in both groups with no change in blood pressure and hence a significant reduction in systemic vascular resistance. The increase in cardiac index occurred as a result of increased heart rate, presumably consequent on both the sympatho-adrenal response to laryngoscopy and intubation and the vagolytic effects of pancuronium. Thereafter blood pressure remained well controlled in both groups, but was lower in the propofol group, and indeed in this group fewer patients required nitroglycerine for perioperative hypertension than in the enflurane group. After sternotomy, systemic vascular resistance was increased in both groups, associated with a decrease in cardiac index and stroke index with heart rate remaining unaltered. At this time our data suggest that the heart was working less efficiently since less work (left ventricular stroke work index) was being done for the same oxygen consumption. This reduction in cardiac work clearly reflects a reduction in flow-related work (decreased cardiac index) rather than in pressure-related work (systemic vascular resistance increased). These observations apply to both groups of patients as a whole, but it is instructive to consider what happens in the individual patient when these factors are exaggerated.

One patient in the enflurane group had a normal blood pressure but a high baseline value of systemic vascular resistance and a low cardiac index. Following sternotomy, systemic vascular resistance was further increased and stroke index significantly reduced. This large increase in systemic vasoconstriction provoked a considerable increase in myocardial oxygen consumption which, in turn, provoked an increase in coronary sinus blood flow as the coronary vasculature dilated in an attempt to increase myocardial oxygen supply and prevent oxygen supply/demand imbalance. Clearly this was not successful as the patient demonstrated lactate production suggestive of anaerobic myocardial metabolism, which occurred with ST segment elevation of 0.5 mm on the ECG (Table 3). Thus, in this patient, severe vasoconstriction and the presence of a low CI, further provoked by autonomic responses to surgery, resulted in the development of ischaemia. The extent to which enflurane anaesthesia can be implicated in the development of ischaemia in this patient is doubtful, although it is possible that an agent with more pronounced systemic vasodilator effects would have proved beneficial. However, we can consider another patient, also in the enflurane group, in whom the administration of enflurane was associated with conversion of anaerobic to aerobic cardiac metabolism. The lactate production and ST segment depression seen after intubation, before enflurane anaesthesia, returned to normal following its introduction. Thus enflurane had beneficial as well as detrimental effects in individual patients.

There were no episodes of lactate production in the patients receiving propofol. There was one episode of regional lactate production, but there were no ECG changes suggestive of ischaemia in this patient. In the propofol group as a whole both myocardial oxygen consumption and coronary vascular resistance were largely unaltered during anaesthesia and surgery. We have no evidence that propofol anaesthesia, in the doses used in this

study, acts as a pharmacological coronary vasodilator and it is therefore unlikely to promote intracoronary steal. However, we do have evidence from individual patients in our study that propofol does not inhibit physiological coronary vasodilatation and thus, where necessary, the coronary circulation is able to increase coronary sinus blood flow in order to maintain myocardial oxygen balance in the presence of increased myocardial oxygen consumption. This is interesting since propofol is well described as having peripheral vasodilator effects resulting in decreased blood pressure [28]. Clearly at the dosage used in this study propofol possesses systemic but not coronary vasodilator effects.

The infusion regimen for propofol used in this study differs from those described elsewhere, both for cardiac [29] and noncardiac surgery [30]. Recently, propofol has been used together with low dose fentanyl to provide anaesthesia for patients prior to myocardial revascularisation [31]. The myocardial depressant effect of propofol was used to control left ventricular work and myocardial oxygen consumption; there was no evidence of anaerobic myocardial metabolism as measured by lactate extraction. The hazards of higher dose infusions in cardiac surgical patients have been described [32] but our regimen achieved haemodynamic stability.

In conclusion, we have shown that fentanyl/propofol anaesthesia does not cause coronary vasodilatation, that its effects are not associated with any abnormality of cardiac metabolism or coronary haemodynamics that would limit its use in patients undergoing coronary artery bypass grafting and that it is comparable to fentanyl/enflurane anaesthesia. These data would support the use of propofol by infusion for maintenance of anaesthesia during cardiac surgery.

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Appendix

Coronary vascular resistance:

$$\text{CVR} = \frac{\text{MAP} - \text{RAP}}{\text{CSBF}} \text{ mmHg.ml}^{-1}.\text{min}^{-1}$$

where MAP = Mean arterial pressure (mmHg)

RAP = Right atrial pressure (mmHg)

CSBF = Coronary sinus blood flow (ml.min⁻¹)

Arterial oxygen content

$$\text{CaO}_2 = (\text{Hb} \times \text{Sat\%} \times 1.39) + (0.003 \times \text{PaO}_2) \text{ ml.dl}^{-1}$$

Myocardial oxygen consumption (MVO₂):

$$\text{Global MVO}_2 = \frac{(\text{CaO}_2 - \text{CcsO}_2)}{100} \times \text{CSBF ml.min}^{-1}$$

$$\text{Regional MVO}_2 = \frac{(\text{CaO}_2 - \text{CqcvO}_2)}{100} \times \text{GCVBF ml.min}^{-1}$$

Where CaO₂ = arterial blood oxygen content (ml.dl⁻¹)

CcsO₂ = coronary sinus blood oxygen content (ml.dl⁻¹)

CgcvO₂ = great cardiac vein blood oxygen content (ml.dl⁻¹)

Lactate extraction ratio:

$$\text{Global Lact Extr} = \frac{\text{CaLact} - \text{CcsLact}}{\text{CaLact}} \times 100\%$$

$$\text{Regional Lact Extr} = \frac{\text{CaLact} - \text{CqcvLact}}{\text{CaLact}} \times 100\%$$

Where CaLact = arterial lactate concentration (mmol.l⁻¹)

CcsLact = coronary sinus lactate concentration (mmol.l⁻¹)

CgcvLact = great cardiac vein lactate concentration (mmol.l⁻¹)

Vital capacity breath technique for rapid anaesthetic induction: comparison of sevoflurane and isoflurane

M. YURINO AND H. KIMURA

Summary

This study compares vital capacity rapid inhalational induction of anaesthesia with sevoflurane and isoflurane. Forty-six volunteers undergoing the procedure had one of the two agents: 25 had sevoflurane and 21 isoflurane. Subjects were unpremedicated and breathed approximately 1.7 MAC equivalents of either vapour. There were no significant differences in the patients' monitored cardiovascular, respiratory, and electrocardiographic variables. The mean time for induction of anaesthesia with sevoflurane (120 s) was significantly shorter than with isoflurane (145 s), reflecting its higher blood–gas solubility. There were fewer induction complications in the sevoflurane group. Subjects in the sevoflurane group found the induction of anaesthesia more pleasant and were more willing to undergo it again compared to subjects in the isoflurane group. We conclude that sevoflurane is superior to isoflurane in vital capacity rapid inhalational induction of anaesthesia, particularly in instances where premedication should be avoided.

Key words

*Anaesthetics volatile; isoflurane, sevoflurane.
Excitatory phase; induction.*

Ruffie and colleagues described the technique of vital capacity rapid inhalational induction (VCRII) in 200 patients [1], who were instructed to take a vital capacity breath of 4% halothane in oxygen, and to hold it in the lungs for 30–90 s until loss of consciousness. The VCRII technique had certain advantages over conventional inhalation and intravenous induction of anaesthesia, including prompt induction without a prolonged excitatory phase and full recovery without 'hangover'. An editorial in *Lancet* acknowledged these advantages, but emphasised the potential for hepatic toxicity in repeated halothane anaesthesia [2].

Two studies have described isoflurane as an alternative to halothane for VCRII [3, 4]. They showed that isoflurane induced loss of consciousness more rapidly than halothane because of its lower blood–gas solubility and that isoflurane was well tolerated by patients. Sevoflurane, on the other hand, has an even lower blood–gas solubility than isoflurane [5], and could be expected to induce anaesthesia more rapidly. In this study, we compared sevoflurane and isoflurane for VCRII.

Methods

The study was approved by the Clinical Human Research Committee, and informed consent was obtained from each

volunteer. Forty-six healthy volunteers were randomly divided into two groups. Twenty-five were assigned to the sevoflurane (S) group and 21 to the isoflurane (I) group, but they were unaware to which group they had been assigned. The groups were demographically similar with no significant differences between them in respect of age, sex, weight, or height. The S group breathed 3% sevoflurane in oxygen, and the I group 2% isoflurane in oxygen. These concentrations represented approximately 1.7 MAC equivalents of each agent. None were premedicated.

Mixtures of each anaesthetic agent together with oxygen were delivered by an Ohmeda Vapor vaporizer into the circle system of an Ohmeda anaesthesia machine. The inspiratory and expiratory limbs of the circle were attached to a Y connector. Respiratory gases were sampled, at a rate of 150 ml.min⁻¹ into the multi-gas monitor (Datex), between the Y connector and the elbow connector with the mask attached to it. First, the mask was replaced by a test lung and the vaporizer was set to a concentration slightly higher than recommended for each inhalation anaesthetic, and the gas in the circuit was circulated. We controlled the vaporizer by monitoring the circulating gas concentration, and then primed the circle according to the desired concentrations. The volunteers, who were breathing room air, were instructed to breathe out to a residual volume.

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Table 1. Demographic data of subjects. Values are expressed as mean (SD) (range).

	Males	Females	Age (years)	Weight (kg)	Height (cm)
3% sevoflurane (<i>n</i> = 25)	19	6	25.9 (3.6) (23–36)	60.6 (8.2) (48–74)	168.9 (7.7) (158–182)
2% isoflurane (<i>n</i> = 21)	16	5	25.8 (4.4) (22–40)	59.1 (11.1) (40–86)	168.1 (9.2) (151–180)

We then applied the anaesthetic system and mask to the volunteers' faces, instructing them to breathe in deeply and to attempt to hold their breath, until they lost consciousness. Following the vital capacity breath, the volunteers were allowed to resume spontaneous respiration of the same anaesthetic mixture for up to 5 min. After that, the subjects continued to breathe oxygen until they regained consciousness.

Loss of consciousness was defined as no response to the command 'open your eyes'. Commands were repeated at 10 s intervals until the subjects failed to respond. Induction time and the presence of excitatory phenomena were recorded. Induction time was defined as the interval between the time at which the subject's lung volume reached total lung capacity (the end of the vital capacity inspiration) and loss of consciousness.

We defined an anaesthetic induction as 'complicated' if one or more complications occurred. We grouped complications into the five categories established by Lamberty and Wilson [3]. A single cough, laryngospasm, breath-holding, movement of a limb, or excessive salivation (defined as enough secretion to wet our hands) were recorded. As it is difficult to assess such observations objectively, we were very strict in our definition. One of the authors acted as observer and evaluated all inductions, while the other anaesthetised the subjects. The observer, who was 'blind' to the anaesthetic agent used, asked the subjects to characterise the smell of the anaesthetic agents and whether they would be willing to undergo VCRII again immediately after emergence from anaesthesia.

We monitored the subjects with an automatic noninvasive blood pressure monitor (Japan, Colin), with an ECG oscilloscope and pulse oximetry. Continuous variables were analysed using one-way analysis of variance (ANOVA) and nonparametric testing. Discrete variables were analysed

using Chi-squared testing. *p* values < 0.05 were considered to be statistically significant.

Results

Anaesthesia was successfully induced in 25 subjects in the S group and in 20 in the I group. One induction in the I group had to be abandoned because the subject displayed severe excitatory movements. The demographic data of the two groups are shown in Table 1.

The mean time for induction of anaesthesia was significantly shorter with sevoflurane than with isoflurane (120 (SD 24) s and 145 (SD 39) s, respectively). Oxygen saturation markers were similar in the two groups. They were about 97% or above before induction and increased slightly after the anaesthetic mask was applied.

In both groups cardiovascular stability was similar, and systolic and diastolic blood pressures gradually decreased, but stayed within safe levels. The differences between the blood pressure values before anaesthesia and at time intervals of 2 to 5 min thereafter were statistically significant in the S group, and at time intervals of 3 to 5 min thereafter were statistically significant in the I group (Table 2). Heart rates did not change significantly (Table 2).

The six most common complications of inhalation induction that occurred in the two groups are presented in Table 3. Overall, the S group had fewer and less severe complications compared to the I group. The gravest complications: coughing, laryngospasm, breath-holding, and excessive salivation did not occur in the S group (Table 3).

Subjects in the S group found the induction more pleasant than did those in the I group and most of them indicated that they would be willing to undergo the same induction again (Table 4).

Table 2. Comparison of cardiovascular stability between anaesthetics. Values are expressed as mean (SD).

	Before induction	After induction, (min)				
	control	1	2	3	4	5
3% sevoflurane (n = 25)						
Systolic blood pressure (mmHg)	124 (17)	122 (15)	118* (13)	113* (14)	108* (12)	104* (11)
Diastolic blood pressure (mmHg)	69 (10)	67 (13)	65* (13)	59* (12)	56* (12)	55* (11)
Heart rate (beat.min ⁻¹)	76 (14)	74 (17)	76 (13)	76 (12)	73 (12)	71 (12)
2% isoflurane (n = 20)						
Systolic blood pressure (mmHg)	117 (13)	118 (16)	114 (13)	111* (14)	109* (13)	107* (13)
Diastolic blood pressure (mmHg)	68 (9)	67 (11)	66 (10)	61* (10)	58* (10)	56* (10)
Heart rate (beat.min ⁻¹)	73 (16)	79 (20)	79 (17)	77 (16)	79 (16)	74 (14)

Control compared with each value *: *p* < 0.05.

Table 3. Incidence of complications during induction of anaesthesia.

	3% sevoflurane (n = 25)	2% isoflurane (n = 21)
<i>Induction</i>		
Complicated	3/25 (12%)*	11/21 (52.4%)
Uncomplicated	22/25 (88%)	10/21 (47.6%)
(1) Coughing	—	9/21 (42.9%)
(2) Larygospasm	—	—
(3) Breath holding	—	1/21 (4.8%)
(4) Movements	3/25 (12%)	9/21 (42.9%)
(5) Secretions	—	—
(6) abandoned	—	1/21 (4.8%)

*significantly different from isoflurane group ($p < 0.01$).

Table 4. Acceptability and smell.

	3% sevoflurane	2% isoflurane
<i>Same induction again?</i>		
yes	20/25 (80.0%)*	8/20 (40.0%)
no comment	1/25 (4.0%)	1/20 (5.0%)
no	4/25 (16.0%)	11/20 (55.0%)
<i>Smell</i>		
pleasant	15/25 (60.0%)**	1/20 (5.0%)
no comment	4/25 (16.0%)	4/20 (20.0%)
unpleasant	6/25 (24.0%)	15/20 (75.0%)

* $p < 0.05$, ** $p < 0.01$.

Discussion

Lamberty and Wilson showed that the VCR II technique with isoflurane was associated with fewer induction complications than a conventional inhalation induction and that isoflurane was a useful alternative to halothane in VCR II [3]. Loper *et al.* also reported that isoflurane could be used as an alternative to halothane for rapid inhalation anaesthesia induction [4].

Although isoflurane does not have the potential for hepatic toxicity associated with halothane, it does cause considerably more complications during conventional inhalational induction [6]. Loper *et al.* compared equipotent vapour concentrations of isoflurane (5%) and halothane (3.5%) in VCR II. To suppress the cough reflex, subjects were premedicated with glycopyrronium and were given fentanyl $5 \mu\text{g.kg}^{-1}$. High concentrations of halothane were well tolerated, but high concentrations of isoflurane were not.

Snider and others also recommended that during VCR II, concentrations of isoflurane higher than 2% should not be used [7]. We therefore used 2% isoflurane in this study, and 3% sevoflurane, since concentrations have approximately the same MAC [8, 9].

The 120 s induction time with sevoflurane was significantly shorter than with isoflurane's 145 s ($p < 0.02$). Nevertheless, these differences were not particularly impressive under clinical conditions. In theory, if the physical characteristics of sevoflurane, isoflurane, and halothane were compared, sevoflurane should have the most rapid induction and emergence, and the least cumulative effect. Loper *et al.* described the mean induction time in VCR II as significantly shorter with 5% isoflurane as compared to 3.5% halothane (38 SD 2) s and 86 (SD 4) s, respectively). If we were able to use higher concentrations

of sevoflurane (7.5%) and isoflurane (5%), the differences in the rapidity of induction would become clinically more apparent, and induction with sevoflurane would probably be more rapid than with isoflurane.

All complications occurred less frequently in the S group than in the I group. Neither coughing nor breath-holding occurred in the S group, but slight finger movements were observed in 12% of the subjects. Due to the pungency of isoflurane, subjects were significantly more prone to coughing and excitatory movements. Small and sometimes excessive movements of the extremities were observed in 43% of the subjects in the I group.

When questioned after emerging from anaesthesia, the S subjects found the VCR II experience more acceptable, with 80% of them willing to undergo VCR II with sevoflurane again. On the other hand, 55% of the subjects in the I group were unwilling to undergo VCR II induction with isoflurane again. Half of the subjects in the I group experienced upper airway irritation great enough to prevent them from being able to hold their breath satisfactorily. Immediately after deep inspiration, they resumed breathing, followed by coughing. Sixty percent of the S group considered the smell of sevoflurane 'pleasant,' while 75% of the I group described the isoflurane smell as 'unpleasant.'

Our findings differ from those of Lamberty and Wilson who found that 94% of subjects well-tolerated the VCR II technique with 2% isoflurane in 67% nitrous oxide and 33% oxygen [3]. Perhaps our criteria for acceptability were more rigorous or the higher percentage of nitrous oxide used by Lamberty's group may have increased their subjects' tolerance [10].

The use of sevoflurane in VCR II and its advantages during recovery from anaesthesia makes it particularly appropriate for same-day and emergency surgeries. In these situations, patients would be unpremedicated, and most able to cooperate. When premedication and intravenous agents can be avoided, patients have the best chance of being alert in the postoperative period.

In conclusion, sevoflurane is superior to isoflurane in VCR II. Its low blood-gas solubility allows for the same, or a more rapid induction than isoflurane. In addition, sevoflurane is associated with fewer complications in the excitatory phase than isoflurane. Subjects tolerate sevoflurane better than isoflurane because it is less pungent and irritating to the upper airways. Sevoflurane may be particularly useful in day care and emergency surgery, and in other cases where premedication and supplementary intravenous agents are contraindicated.

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Upper oesophageal sphincter pressure during inhalational anaesthesia

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Summary

Upper oesophageal sphincter pressure was recorded with a Dent sleeve in 30 patients breathing nitrous oxide, oxygen and halothane. Twenty-three patients, after thiopentone induction, received suxamethonium and had their trachea intubated either before (group A, $n = 11$), or after (group B, $n = 11$), a study period of inhalational anaesthesia. Group C ($n = 8$) received an inhalational induction. Mean (SD) sphincter pressure after loss of consciousness was 8 (7) mmHg (group A), 6 (5) mmHg (group B) and 24 (13) mmHg (group C) increasing to 19 (7) mmHg in group A immediately after intubation. With an end-tidal halothane concentration of 1.5%, mean sphincter pressure in group B, 16 (7) mmHg, was significantly lower than in group A, 45 (21) mmHg ($p < 0.001$) and group C, 27 (14) mmHg ($p < 0.05$). Halothane had no dose-related effect on sphincter pressure. Insertion of a laryngeal mask in group C ($n = 7$) had no significant effect on sphincter pressure. Induction and maintenance of anaesthesia with halothane, unlike thiopentone or suxamethonium, maintained a degree of upper oesophageal sphincter tone, although three patients in this study had sphincter pressures of less than 10 mmHg and would therefore have been at risk of regurgitation in the presence of gastro-oesophageal reflux.

Key words

Gastrointestinal tract; upper oesophageal sphincter.

Complications; regurgitation.

Anaesthesia; inhalational.

Gastro-oesophageal reflux in conscious patients usually raises oesophageal pressure by less than 10 mmHg [1] and the upper oesophageal sphincter normally prevents regurgitation of oesophageal contents into the pharynx. Upper oesophageal sphincter pressure (UOSP) is usually about 40 mmHg but is not constant and varies with the state of arousal [2]; increasing with mental stress [3] and decreasing to a mean of 8 mmHg during deep sleep [4].

We have previously reported [5] that sedation with midazolam, induction of anaesthesia with thiopentone and neuromuscular blockade with suxamethonium can all decrease UOSP to below 10 mmHg, a level that would allow regurgitation in most patients with gastro-oesophageal reflux. However, during light general anaesthesia without muscle relaxants the upper oesophageal sphincter can prevent the regurgitation of oesophageal contents [6, 7]. Studies of UOSP during inhalational anaesthesia have not previously been reported.

In the present investigation we have measured UOSP continuously in patients breathing nitrous oxide and oxygen with increasing concentrations of halothane through three different airways. This investigation is part of

a larger study of UOSP during anaesthesia. Two other aspects of this study, in which some of the patients in the present investigation also took part, have already been reported [5, 8].

Method

Measurement of UOSP

Continuous UOSP was measured by a manometry catheter with a Dent sleeve [9]. The sleeve is a 6 cm long collapsible rubber membrane which, when perfused with water, records the highest pressure applied along its length. Oesophageal pressure was also recorded through a sidehole at the distal end of the sleeve. The catheter and the method of positioning it in the upper oesophageal sphincter have been described previously [5, 8]. The catheter was zeroed to a level behind the cricoid cartilage before insertion.

The procedure

Hospital ethics committee approval was obtained and 31 patients from ASA categories 1 and 2, scheduled for elec-

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Table 1. Group allocation, induction and airway.

	Group A	Group B	Group C
<i>n</i>	12	11	8
Induction	Thiopentone	Thiopentone	Inhalational
Airway	Tracheal tube	Guedel airway	Guedel airway
Second airway	—	—	Laryngeal mask

tive surgery, in whom there was no contraindication to halothane, gave informed consent. One hour after pre-medication with intramuscular papaveretum and hyoscine, and shortly before surgery, the manometry catheter was swallowed via an anaesthetised nostril. The patient then lay on a standard pillow of 6.0 cm of firm foam rubber with an integral firm neck support to hold the head and neck in a standard position and 2 min later the UOSP was recorded while the patient was awake.

The patients were divided into one of three groups (Table 1). In the first 23 patients anaesthesia was induced with thiopentone 4 mg.kg^{-1} and UOSP was recorded 30 s after loss of consciousness. These patients were then randomly allocated to receive suxamethonium and undergo tracheal intubation either before (group A, $n = 12$) or after (group B, $n = 11$) the administration of inhalational anaesthesia. Group C ($n = 8$) received an inhalational induction with nitrous oxide 67% and oxygen 33% for 2 min, followed by the addition of increasing concentrations of halothane; UOSP was recorded at loss of the eyelash reflex.

Group A were given suxamethonium 1 mg.kg^{-1} and their tracheas intubated with a cuffed PVC tube, size 8.0 mm internal diameter for females and 9.0 mm for males; UOSP was recorded before and after intubation. A Guedel oral airway with a facemask was used in groups B and C, size 2 in females and 3 in males. Anaesthesia was maintained in all groups by spontaneous breathing through a Magill attachment of nitrous oxide 4 l.min^{-1} and oxygen 2 l.min^{-1} with varying concentrations of inspired halothane. Gas was sampled by a catheter inserted 4 cm into the oral airway in groups B and C, and by a T-connector on the tracheal tube in group A. End-tidal halothane concentration was measured by a Datex Capnomac which was calibrated before each patient. In all groups UOSP was then recorded at incremental levels of end-tidal halothane concentration between 0.8% and 1.8% (between 0.8% and 1.5% in group C), which was increased in a standard manner by setting the inspired concentration to 1% above the desired end-tidal concentration. The higher levels were not pursued if hypotension occurred. In groups A and B the first readings were taken between 8 and 10 min after the injection of thiopentone.

Groups B and C were given suxamethonium 1 mg.kg^{-1}

and UOSP was recorded 1 min later. Group B patients were then intubated as above and, together with group A, had completed the study. Group C patients had a laryngeal mask airway (LMA) inserted, size 3 for females and size 4 for males. After inflation of the cuff (20 ml for females, 30 ml for males), the LMA was allowed to find its own level, as described by Brain. UOSP was then measured with the patient still paralysed and the laryngeal mask airway in place. Gas was sampled by a T-connector on the LMA. Five minutes after the return of adequate spontaneous breathing through the LMA, the UOSP was recorded with an end-tidal halothane concentration of 1%. End-tidal carbon dioxide concentration ($\text{P}_{\text{ET}}\text{CO}_2$) was measured in group C at 1% end-tidal halothane concentration with both the Guedel airway and the LMA. The LMA was marked at the incisors and the distance to the tip was measured after removal.

Statistics

The slopes of UOSP against end-tidal halothane concentration in each patient varied within each group so that the dose-related effect in each group was assessed by the mean of these slopes, and compared between groups by the analysis of variance of these means. As the mean slopes varied between groups, UOSP could only be compared between the groups at one end-tidal halothane concentration: group A was compared with group B and group B with group C at an end-tidal halothane concentration of 1.5% by the unpaired *t*-test on the $\log_{10}$ data (which decreased the difference in variation between the groups). Other changes between groups were also analysed by the unpaired *t*-test. Changes of UOSP within each group were analysed by the paired *t*-test. Statistical significance was assumed at the 5% level.

Results

The sleeve pressure was always higher than oesophageal pressure, the latter usually varied between 0 and 5 mmHg; the sleeve pressure therefore represented UOSP and not oesophageal pressure. One patient in group A coughed and would not breathe with the tracheal tube in place and was therefore paralysed and the lungs ventilated and not studied. One patient in group C developed laryngospasm after insertion of the LMA and was also paralysed, intubated and the lungs ventilated; this patient's results up until this point are included. Patient details in the three groups are similar and are shown in Table 2.

UOSP with increments of end-tidal halothane concentration are shown in each patient in the three groups in Figure 1. UOSP in each patient in group C during inhalational induction of anaesthesia is shown in Figure 2. Mean UOSP

Table 2. Patient details, mean (range).

	Group A	Group B	Group C
<i>n</i>	11	11	8
Females	6	6	4
Premedicated	9	8	6
Papaveretum; mg.kg^{-1}	0.18 (0.14–0.23)	0.19 (0.15–0.27)	0.18 (0.14–0.20)
Age; years	48 (37–56)	47 (33–64)	45 (25–58)
Weight; kg	71 (52–100)	69 (55–110)	72 (55–90)

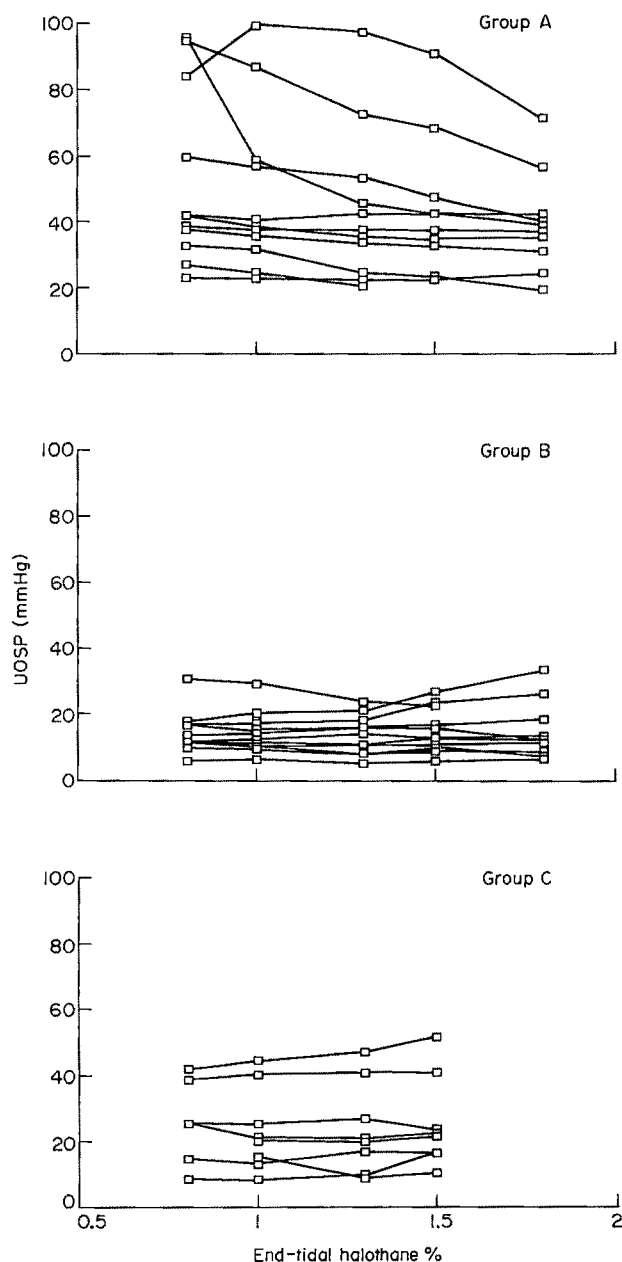


Fig. 1. Upper oesophageal sphincter pressure (UOSP) in mmHg in each patient, in the three groups, while breathing increasing concentrations of halothane.

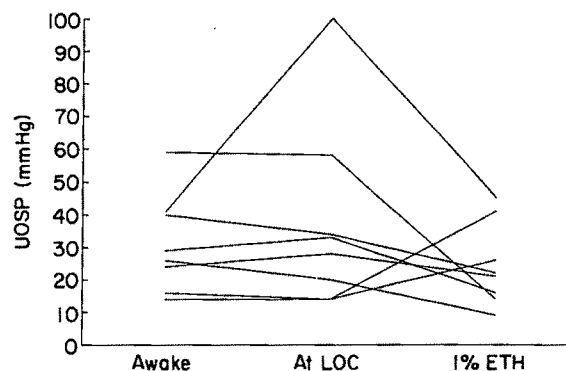


Fig. 2. Upper oesophageal sphincter pressure (UOSP) awake, at loss of consciousness (LOC) and with an end-tidal halothane concentration of 1% (1% ETH) during the inhalational induction of anaesthesia in each patient in group C.

in the three groups at each stage of the study is shown in Table 3 and in the sequence in which they were measured in Figure 3.

In group B, mean UOSP 30 s after thiopentone was no different than after suxamethonium. Ten minutes after thiopentone, mean UOSP with 1.5% end-tidal halothane concentration was significantly higher than after suxamethonium ($p < 0.01$); however, it was still lower than in group C at 1.5% end-tidal halothane concentration ($p < 0.05$) in whom thiopentone had not been given. In groups B and C there was no dose-related effect of halothane on UOSP; the mean of the slopes of UOSP against end-tidal halothane concentration was $+2.4 \text{ mmHg.\% end-tidal halothane concentration}^{-1}$ in group B and $+2.6 \text{ mmHg.\% end-tidal halothane concentration}^{-1}$ in group C (Fig. 1).

In group A, the mean UOSP after suxamethonium was increased by 12 (SD 7) mmHg after intubation (Table 3) and the mean UOSP immediately after intubation was the same as that after vecuronium 0.1 mg.kg^{-1} over 15 min later when the study was completed. Mean UOSP at 1.5% end-tidal halothane concentration was significantly higher than in group B at 1.5% end-tidal halothane concentration ($p < 0.001$). However, when the effect of the tracheal tube was subtracted from each value of UOSP in group A there was no significant difference from group B at 1.5% end-tidal halothane concentration ($p = 0.11$). The mean of the slopes of UOSP against end-tidal halothane concentration

Table 3. Upper oesophageal sphincter pressure, mean (SD) (range).

	Group A	Group B	Group C
<i>n</i>	11	11	8
Awake	45 (24) (18–89)	38 (18) (8–75)	31 (15) (14–59)
Thiopentone	8 (7) (0–17)	6 (5) (1–21)	—
0.8% ETH	53 (27) (23–96)	15 (6) (6–31)	26 (13) (9–42)
1.0% ETH	49 (25) (23–101)	15 (6) (7–30)	24 (13) (9–45)
1.3% ETH	45 (23) (21–98)	15 (6) (6–22)	25 (14) (10–48)
1.5% ETH	45 (21) (23–91)	16 (7) (7–28)	27 (14) (12–53)
1.8% ETH	40 (15) (20–72)	17 (9) (8–35)	—
Suxamethonium	7 (6) (1–20)	8 (4) (3–19)	7 (4) (4–15)
After intubation	19 (7) (9–30)	—	—
After LMA ($n = 7$)	—	—	7 (5) (1–16)
1.0% ETH with LMA ($n = 7$)	—	—	17 (10) (9–39)

ETH, end-tidal halothane concentration; LMA, laryngeal mask airway.

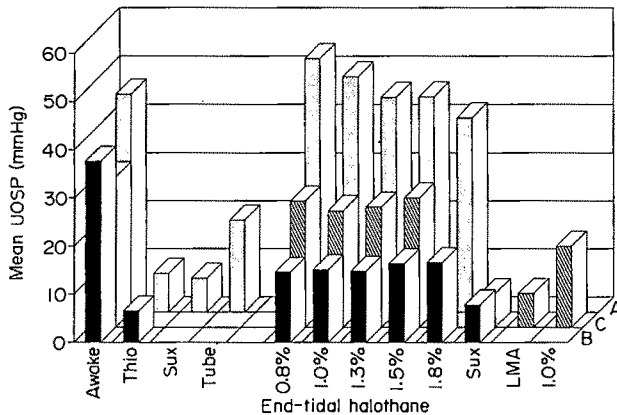


Fig. 3. Mean upper oesophageal sphincter pressure (UOSP) at each stage of the investigation in the three groups A, B and C for comparison. Thio, thiopentone; Sux, suxamethonium; Tube, tracheal tube; LMA, laryngeal mask airway.

in group A was $-14 \text{ mmHg} \cdot \% \text{end-tidal halothane concentration}^{-1}$ (Fig. 1) which was significantly different from the mean of the slopes in the other two groups ($p < 0.01$).

During inhalational induction in group C, when 67% nitrous oxide was given for 2 min before halothane, there was no change in UOSP. When halothane was added, UOSP did not decrease until after loss of consciousness (Fig. 2). UOSP at 1% end-tidal halothane concentration was not significantly lower than the awake value ($p = 0.4$) and insertion of the LMA and inflation of the cuff did not alter the residual UOSP after suxamethonium (Table 3). The distance from the tip of LMA to the incisors (mean 16.5 cm) was always at least 2 cm less than the distance of the proximal sleeve of the manometry catheter from the nares (mean 19 cm), and therefore the LMA did not encroach on the pressure sensing sleeve. Mean UOSP at 1% end-tidal halothane concentration with the LMA ($n = 7$) was significantly higher than after suxamethonium ($p < 0.01$) and lower but not significantly different from mean UOSP at 1% end-tidal halothane concentration with the Guedel airway ($p = 0.47$). However, there was a wide 95% confidence interval of the difference between the means of -9 to 23 mmHg . Mean $\text{Fe}'\text{CO}_2$ when breathing through the LMA was 6.9 ($\text{SD} = 1.1$)% at 1% end-tidal halothane concentration, which was significantly higher than mean $\text{Fe}'\text{CO}_2$ of 6.2 ($\text{SD} = 0.6$)% when breathing through the

pentone or suxamethonium was therefore usually low enough to allow regurgitation in the presence of gastro-oesophageal reflux. In general, inhalational anaesthesia maintained a higher UOSP than thiopentone or suxamethonium, although at 1% end-tidal halothane concentration, UOSP was less than 10 mmHg in three patients: one from group B (7 mmHg), one from group C with a facemask and Guedel airway (9 mmHg) and one from group C with a LMA (9 mmHg). These three patients would have regurgitated if oesophageal contents at a pressure of 10 mmHg had been present. These findings can explain those of Blitt *et al.* [11] who showed that the regurgitation of dye into the pharynx occurred during 15% of 240 general anaesthetics with tubocurarine compared to 3.5% of 194 general anaesthetics without neuromuscular blocking drugs.

There was no dose-related effect of halothane on UOSP in groups B or C. Nitrous oxide without halothane did not appear to have an effect on UOSP. The changes in UOSP with inhalational anaesthesia are therefore more likely to be due to changes relating to the state of unconsciousness than to the drugs themselves. The function of pharyngeal or upper airway muscles has not previously been studied during inhalational anaesthesia in humans. In the cat, Nishino *et al.* [12] showed a dose-dependent decrease in activity of the hypoglossal and phrenic nerves with halothane, but the recurrent laryngeal nerve retained its activity during deep halothane anaesthesia. However, the motor nerve supply of cricopharyngeus in man is from none of these nerves, it is probably from the pharyngeal branch of the vagus via the pharyngeal plexus [13].

We have previously described an increase in UOSP with a tracheal tube [8] caused by a posterior force developed when the tube is flexed to conform to the shape of the airway. The decrease in UOSP with increasing end-tidal halothane concentration in group A could possibly have been explained by the contribution from the tracheal tube decreasing as it warmed to body temperature [14], but the mean UOSP immediately after intubation with suxamethonium was the same as that after vecuronium over 15 min later when the study was completed. Alternatively, therefore, this decrease in UOSP with increasing end-tidal halothane concentration might be caused by the gradual offset of a reflex increase in the muscle tone of cricopharyngeus from the central stimulation of laryngoscopy and intubation.

During inhalational induction in group C, UOSP was well maintained until after loss of consciousness. This is in contrast to intravenous induction with thiopentone [5]

normal gastric pressures. UOSP is occasionally below this level with either a Guedel airway or LMA and regurgitation may then occur.

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Peri-operative liver graft function: monitoring using the relationship between blood glucose and oxygen consumption during anaesthesia

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Summary

The purpose of our study was to test the hypothesis that the quotient between plasma glucose and whole body oxygen consumption ($\dot{V}O_2$) as a 'metabolic index' is a sensitive indicator of early graft function. Arterial levels of glucose and oxygen consumption were determined in 100 consecutive patients during orthotopic liver transplantation performed without anhepatic veno-venous bypass. Patients were divided into survivors with no obvious problems related to graft function and those with primary nonfunction of the graft. The neohepatic increase in $\dot{V}O_2$ was significantly higher in survivors (112 ± 4 vs 88 ± 11 ml.min⁻¹.m⁻²; $p < 0.05$), whereas blood glucose levels after reperfusion were higher (352 ± 18 vs. 287 ± 36 mg dl⁻¹) in those with primary non-function of the graft. The calculated metabolic index was also higher (4.02 ± 0.93 vs 2.67 ± 0.45 , $p < 0.05$) in patients with primary nonfunction of the graft. Our principal conclusion was that 92% of normal functioning liver grafts could be classified correctly by the metabolic index immediately after reperfusion, whereas glucose levels and $\dot{V}O_2$ alone classified only 67% and 70% of normal functioning liver grafts correctly.

Key words

Surgery; transplantation, liver.

Metabolism; glucose.

Oxygen; consumption.

Orthotopic liver transplantation (OLT) has become important in the management of patients with acute hepatic failure and is now used with remarkable success in order to treat end-stage liver failure. An early assessment of liver graft function is important to determine outcome, because the decision to retransplant has to be made rapidly when the graft fails. Continuous monitoring of hepatic allograft function during the neohepatic stage of reperfusion could be useful clinically. As discussed previously by Fath and colleagues [1] most routine liver function tests fail to indicate promptly the actual metabolic capacity of the newly grafted organ. An immediate indicator of liver graft function could help in the timing for early re-transplantation. Both the oxygen consumption index [2] and reperfusion hyperglycaemia [3] have been reported as reliable predictors of subsequent graft function.

In our study we calculated the quotient of blood glucose levels and total body oxygen consumption ($\dot{V}O_2$). We determined whether this calculated 'metabolic index' could serve as a sensitive indicator of early graft function or failure and tested if, in combination, these two parameters were more sensitive in gauging functional recovery of the grafted liver, compared to blood glucose or $\dot{V}O_2$ alone.

Patients and methods

After Hospital Ethics Committee approval and patient consent, orthotopic liver transplantations were performed in 100 consecutive patients at the Vienna Transplantation Centre. The patients (45 female and 55 male) were aged 47 (SD 12) years. The indications for transplantation consisted of chronic active hepatitis in cirrhotics in 39, primary biliary cirrhosis in 31, tumour in cirrhosis in 14 and secondary liver tumours in 16 patients. According to the graft viability, we defined as group 1 patients with normal liver graft function and a survival time of more than 3 months (survivors, $n = 90$), as group 2, 10 patients with primary nonfunction of the graft (PRNF). PRNF was defined by the inability to correct coagulopathy by routine replacement of blood components, distinct increase of liver enzymes, haemodynamic instability and nonsurgical bleeding with subsequent retransplantation within 24 h.

Anaesthesia was induced with intravenous thiopentone (3–5 mg.kg⁻¹), fentanyl (100–200 µg) and diazepam (10 mg); muscle relaxation was achieved with pancuronium (4–8 mg). Volume-cycled ventilation was established using a mixture of oxygen in air and isoflurane with a

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positive end-expiratory pressure of 5–6 mmHg to keep the arterial PO_2 above 100 mmHg and the arterial PCO_2 around 35 mmHg (Servo 900 C-Ventilator, Siemens). To maintain body temperature, all patients were positioned on a heating blanket set at 38°C. The inspired gases were warmed and humidified using a dual servo-heated humidifier (MR 450 Fisher and Paykel). Subsequent boluses of opioids and muscle relaxants were administered as required.

Haemoglobin and haematocrit levels were corrected by replacing packed red cells. Cristalloid and glucose-free solutions were infused to maintain renal perfusion and function. While transplantation was taking place patients received dexamethasone (40 mg bolus during reperfusion), dopamine ($3 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) and mannitol (100 ml 20%) during the anhepatic phase for renal protection.

Measurements and blood samples were taken at the following times: after induction of anaesthesia A, control; before, B; and after crossclamping, C; at the end of the anhepatic period, D; during, E; and after reperfusion, F. Blood glucose concentrations (glucose analyser, Beckmann instruments), arterial and mixed venous oxygen content (CO-oximeter OSM3 Radiometer Copenhagen) and saturation, arterial and mixed venous acid base-state, haemoglobin and haematocrit were measured. The donor organs were preserved using the modified solution of the University of Wisconsin. The surgical procedure was subdivided into three phases. Phase I was defined as the time interval from induction of anaesthesia to removal of the recipient liver, phase II was the anhepatic phase and finally, phase III the period between the unclamping of the caval vessels and the end of surgery. No anhepatic veno-venous bypass was used in any patient. The grafted liver was reperfused at the beginning of phase three by unclamping in sequence the portal vein then the supra- and infrahepatic vena cava inferior. No recipients received any glucose during the procedure. Total body oxygen consumption ($\dot{V}O_2$) was calculated according to the formula: $\dot{V}O_2 = \text{cardiac index} \times \text{arteriovenous oxygen content difference}$. Body temperature was measured with a thermal probe in

the pulmonary artery catheter. At each time point (A–F), cardiac output was measured by thermodilution, and oxygen delivery was calculated as the product of cardiac output and arterial oxygen content.

All data are presented as mean (SD). Data were analysed using analysis of variance and Tukey's method for multiple comparisons. $p < 0.05$ was considered statistically significant. The metabolic index was calculated for each recipient as quotient of blood glucose and oxygen consumption. In addition, changes in metabolic index, blood glucose and oxygen consumption during the neohepatic period were examined, using discriminant analysis to calculate sensitivity, specificity and the positive and negative predictive values.

Results

According to either the presence or absence of initial postoperative graft function, the patients were divided into two groups i.e. graft function ($n = 90$) and peri-operative graft nonfunction ($n = 10$). There was no significant difference in age (survivors; years, mean (SD): 48 (14) vs PRNF: 52 (12)) and anhepatic duration (survivors; min, mean (SD): 80 (16) vs PRNF: 87 (23)). Sixteen patients with sufficient initial graft function died within 12 months through sepsis and multiple organ failure. However, in the patients requiring retransplantation, six survived more than 8 months and four died after multiple organ failure.

Haemodynamic changes

During surgery, in all patients cardiovascular function was relatively stable (mean arterial pressure > 70 mmHg, heart rate < 100 beats.min⁻¹) except during cross-clamping of the vessels, which was characterized by an increase in heart rate > 110 , mean arterial pressures between 55 and 65 mmHg and a profound decrease in cardiac index (CI) associated with decreasing oxygen delivery in both groups. After reperfusion of the graft, both groups showed significant increases in CI (Table 1).

Table 1. Haemodynamic and metabolic changes during orthotopic liver transplantation.

Phase	A	B	C	D	E	F
Metabolic index; $\text{mg} \cdot \text{dl}^{-1} \cdot \text{min} \cdot \text{m}^2 \cdot \text{ml}^{-1}$						
(1)	1.25 (0.24)	1.81 (0.05)	2.26 (0.19)§	1.92 (0.21)§	2.67 (0.45)§*	2.59 (0.51)§*
(2)	1.44 (0.34)	1.93 (0.42)	2.21 (0.45)§	2.41 (0.68)§	4.02 (0.93)§¶*	4.35 (0.36)§¶
Blood temperature; °C						
(1)	36.1 (0.1)	5.8 (0.3)	35.4 (0.6)	35.0 (0.6)§	34.5 (0.5)§	35.2 (0.1)§*
(2)	36.0 (0.2)	35.7 (0.2)	35.3 (0.2)	35.0 (0.3)§	34.1 (0.3)§¶	34.3 (0.3)§¶*
Oxygen delivery; $\text{ml} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$						
(1)	710 (31)	706 (40)	344 (19)§	363 (19)§	868 (41)§¶	746 (35)
(2)	665 (67)	704 (111)	314 (56)§	430 (77)§	754 (88)¶	679 (64)
CI; $\text{l} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$						
(1)	4.6 (0.2)	4.5 (0.2)	2.0 (0.1)§	2.0 (0.1)§	5.2 (0.2)¶	4.6 (0.1)
(2)	3.8 (0.6)	4.6 (0.6)	1.9 (0.3)§	2.2 (0.4)§	4.4 (0.4)¶	4.2 (0.4)
$\dot{V}O_2$; $\text{ml} \cdot \text{min}^{-1} \cdot \text{m}^{-2}$						
(1)	92 (7)	98 (19)	81 (7)	76 (3)§	112 (4)¶*	125(12)¶*
(2)	93 (8)	91 (8)	77 (8)	86 (10)	88 (11*)	83 (6)*
Arterial blood glucose; $\text{m} \cdot \text{mol} \cdot \text{l}^{-1}$						
(1)	5.9 (0.94)	8.9 (3.2)	9.0 (0.94)	7.3 (1.4)	15.9 (2.0)	13.4 (0.6)
(2)	6.6 (0.78)	8.2 (1.1)	8.0 (0.6)	9.6 (2.4)	19.5 (1.0)	18.5 (2.7)

Peri-operative changes in haemodynamic and metabolic parameters in survivors (1) and patients with PRNF (2). (Values are expressed as mean (SD)). A, control after induction of anaesthesia; B, pre-anhepatic; C, after cross-clamping; D, before unclamping; E, after unclamping; F, after reperfusion.

MI, metabolic index; CI, cardiac index; § = significantly different from the pre-anhepatic control (A), $p < 0.05$. * = significantly different between groups, $p < 0.05$, ¶ = significantly different from the anhepatic stage D, $p < 0.05$.

Table 2. Discriminant analysis of variables at timepoint 5 (reperfusion).

Variable	True and false negative	False and true positive	Correctly classified
Blood glucose			
Group 1	67.1%	32.9%	67.4%
Group 2	30%	70%	
$\dot{V}O_2$			
Group 1	69.5%	30.5%	70.6%
Group 2	20%	80%	
Metabolic index			
Group 1	96.3%	3.7%	92.4%
Group 2	40%	60%	

Group 1, survivors; group 2, primary nonfunction; $\dot{V}O_2$, oxygen consumption.

Changes in oxygen delivery and consumption

For oxygen delivery in both groups, a similar pattern in survivors and PRNF was observed during surgery (Table 1). Whole body oxygen consumption was not statistically different between groups, either initially or during cross-clamping. After unclamping of the caval vessels and after reperfusion of the graft, oxygen consumption increased in survivors significantly over the pre-anhepatic control values (112 (4) and 125 (12) respectively, $p < 0.05$) whereas in patients with subsequent graft failure, $\dot{V}O_2$ decreased compared to pre-anhepatic values (88 (11) and 83 (6) respectively).

Metabolic changes

The peri-operative changes in blood temperature, blood glucose and metabolic index are also presented in Table 1. During phases 1 and 2 of transplantation there was no significant difference in plasma glucose concentration in either groups. Immediately after reperfusion (E), glucose levels rose to 287 (36) in survivors and 352 (18) mg.dl⁻¹ in patients with subsequent graft failure ($p < 0.001$). After reperfusion (F), PRNF patients still showed higher levels of blood glucose (333 (48) mg.dl⁻¹).

The calculated metabolic index did not change significantly during the pre-anhepatic and anhepatic period in survivors and PRNF patients, but it rose in both groups compared to control (A). However, after reperfusion, patients with PRNF showed significantly higher values (4.02 (0.93) and 4.35 (0.36)) than survivors (2.67 (0.45) and 2.59 (0.51), $p < 0.05$, Table 1).

Discriminant analysis

Table 2 shows discriminant values of metabolic index during recovery in survivors and PRNF patients. By focusing on the first measurements after reperfusion of the graft, 92.4% of patients were classified correctly. Of survivors 96.3% were classified as true negative and 3.7% as false positives. For PRNF patients, 60% were classified as true positives and 40% as false negatives using the metabolic index as crucial value. At the same timepoint, discriminant analysis of plasma glucose classified 67.1% of survivors as true negatives and 32.9% as false positives, whereas PRNF was classified as 70% true positives and 30% false negatives. Finally, analysis of $\dot{V}O_2$ discriminates 69.5% of survivors as true negatives and 30.5% as false positives; as regards PRNF, 20% were false negatives and 80% true positives. After unclamping, using the metabolic

index as an discriminator, the sensitivity was 95%, the specificity 67% with a prevalence of disease of 90% and an accuracy of 92%.

Discussion

The present study examined the relationship between plasma glucose levels and changes in whole body oxygen consumption during orthotopic liver transplantation. The principal finding is a more accurate predictability of neohepatic recovery based on the metabolic index. This was confirmed by means of a stepwise discriminant analysis of glucose and oxygen consumption and the ratio between these variables.

Although experimental data [4,5] indicate an important effect of acute and chronic hypoxia on carbohydrate metabolism, the possible impact of oxygen supply and consumption with respect to blood glucose metabolism under clinical conditions has not been investigated during liver transplantation in man. Therefore OLT may be a clinical model, which offers the possibility of studying the role of the liver in glucose metabolism under a variety of conditions, including anaesthesia, and dramatic changes in haemodynamic and metabolic conditions.

Blood glucose kinetics in patients undergoing OLT are reported to differ. Hypoglycaemia was described in the absence of the liver [6–8] and the authors suggested that glucose should be given in sufficient quantity so that hypoglycaemia does not occur, especially during the postanhepatic procedure. In contrast, other recent studies by Mallet *et al.* and de Wolf *et al.* [3,9] demonstrated that hypoglycaemia is not a major concern during OLT, but that a tendency to hyperglycaemia occurred as a consequence of anaesthesia and surgical stress. The discrepancy between these studies could be the result of differences in the inclusion criteria for patient selection and different peri-operative surgical and anaesthetic management. Changes in blood glucose levels can be also attributed to a loss of organ function, which might be due either to parenchymal injury or a decrease in hepatic oxygen supply [10, 11].

Our data on peri-operative plasma glucose changes are in agreement with those previously reported by Mallet and coworkers [3]. In both survivors and PRNF patients, significant increases in glucose levels occurred in two steps during the procedure: from control (A) to anhepatic (C) and from anhepatic (D) to neohepatic stage (E). However, by focusing on the reperfusion phase (E,F) only 67% of patients are classified correctly using blood glucose levels alone.

In our study, $\dot{V}O_2$ showed a different pattern in surviving patients than in those with primary graft dysfunction. After unclamping and during reperfusion of the graft, survivors show significantly higher $\dot{V}O_2$ values, as compared to control and to the PRNF group. However, by focusing on sensitivity and specificity, these differences appear to be less important for true classifications of graft viability, as only 70% of patients can be classified accurately. These variations in $\dot{V}O_2$ lead to important questions about measuring or calculating oxygen consumption. In our study, oxygen consumption is commonly derived by Fick's principle and $\dot{V}O_2$ is calculated from cardiac index and measured oxygen content difference. This method has been shown to be in good agreement with results achieved by the direct measurements of respiratory gas exchange [12]. In addition, the multiple sources of error are potentially magnified in the critically ill patient who requires supplementary oxygen and mechanical ventilation and who suffers from liver failure [13].

The different oxygen consumption pattern in survivors and PRNF patients could well parallel the peri-operative blood glucose levels, which are much higher in patients with low oxygen consumption who subsequently develop PRNF. This inverse relationship of $\dot{V}O_2$ and blood glucose of patients undergoing OLT is substantiated by similar findings in animal models of septic shock [14], and in a variety of other states associated with an increased oxygen debt [15]. An additional explanation involves the denervation of the portal vessels, which has been shown to interfere with liver glucose uptake and gluconeogenesis [16].

Clinical limitations of this study are excessive surgical bleeding with higher amounts of donor blood, a substantial decrease in body temperature or the necessity of high therapeutic dosage with catecholamines. However, the immediate increase in plasma glucose (more than 100%) in our groups cannot be explained solely by the flushing of preservation solution, blood supply and administration of dexamethasone. Moreover, glucose intolerance, which is observed in humans after massive liver resection [17], has been found to be closely related to the decrease in hepatic energy charge and the derangement of mitochondrial function [18]. This would explain the relatively good correlation of the lack of an increase in oxygen consumption of the new liver and the simultaneous rise in blood glucose in patients with impaired graft function.

In conclusion, better predictability of new liver recovery, based on the metabolic index, is confirmed by a stepwise discriminant analysis of glucose, oxygen consumption and their ratio. Therefore, we consider it necessary to take both blood glucose and whole body oxygen consumption into account to evaluate the metabolic function of the new liver. Despite possible errors in clinical measurement, these different patterns may be important for the surgical and anaesthetic management of patients undergoing orthotopic liver transplantation. During reperfusion of the liver, patients with normal graft function showed a significantly lower metabolic index than the group with early nonfunction of the liver graft. However, the predictability for postoperative graft failure was lower than for normal graft function. This may be because the PRNF group was smaller. Although survival is influenced by many factors besides initial graft function, the onset of the metabolic function during recovery of the new liver plays a pertinent role in survival. In our population, liver graft function

could be successfully discriminated by this new metabolic index.

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Comparison of digital blood pressure, plethysmography, and the modified Allen's test as means of evaluating the collateral circulation to the hand

T. M. FUHRMAN, T. E. REILLEY AND W. D. PIPPIN

Summary

The collateral circulation to the hand was evaluated on 70 hands of healthy volunteers. Comparisons were made between the results of the modified Allen's test alone and the Allen's test combined with either plethysmography or digital blood pressure. The modified Allen's test requires patient cooperation and the results can be subjective. Plethysmography does not require patient cooperation and produces a signal that varies directly with flow; however, this is not a quantifiable signal. Digital blood pressure (measured by the 2300 Finapres noninvasive blood pressure monitor, Ohmeda, Englewood, CO, USA) also requires no patient cooperation. The values produced are of clinical value and reproducible. Both the plethysmograph and digital blood pressure monitors were able to demonstrate the dominant arterial vessel of the hand. The digital blood pressure monitor produces an objective recordable numerical value, an accepted clinical parameter, and it does not require patient cooperation. The use of a digital blood pressure monitor may prove to be an acceptable alternative to the traditional Allen's test.

Key words

Arteries; Allen's test.

Measurement techniques; oximetry.

Equipment; Finapres.

It has been over 60 years since E.V. Allen described a method for evaluating thromboangiitis obliterans [1]. This test was later modified [2] and was for many years the basic method of assessing the collateral circulation to the hand. Since then many other tests have been used instead of the Allen's test [3–9]. Pulse oximetry has been found to be of limited usefulness [7, 9], however, several studies do suggest its use [4, 5, 8]. Oximetry requires no patient cooperation, but significant variations in flow do not always result in a change of saturation [10]. Recently, we tested plethysmography as a method of evaluating collateral circulation [7]. This also requires no patient cooperation, but the signal varies directly with flow [11–13] and with the addition of a recorder can produce a hard copy for documentation. However, the waveform is not quantifiable, and many monitors have an auto-gain feature, which automatically enlarges the waveform displayed if a reduction in the pulse strength is sensed. Digital blood pressures have been studied in the past in order to assess arterial insufficiency or obstruction [14, 15]. In 1981 Husum and Berthelsen studied the systolic pressure in the thumb in comparison with the Allen's test [16]. The methodology involved was felt to be too complex to replace the Allen's test. However, digital blood pressure measurement is now clinically feasible [17].

This protocol was designed to study the use of digital blood pressure to evaluate the collateral circulation to the hand. The ideal method of evaluation would be noninvasive, nonsubjective, reproducible, provide a quantifiable result and not require patient cooperation.

Materials and Methods

This study was approved by our Institutional Review Board and, after informed consent, 70 hands of healthy volunteers were examined. The three tests were performed in a well-lit room in random order. The modified Allen's test was performed by having the subject open and tightly close their hand while the ulnar and radial arteries were occluded by one of the investigators. After several cycles of opening and closing, the subject opened the hand having been instructed not to hyperextend the fingers [18]. At that point the investigator released one of the arteries and the time from the release of occlusion to the return of colour to the palm of the hand was recorded. This was then repeated with release of the other artery.

The second test involved placing a plethysmographic probe on the index finger, and the arteries were occluded one at a time. The gain on the monitor was adjusted to

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Fig. 1. Digital blood pressure and plethysmograph waveform of a subject with an incomplete palmar arch as noted by blood pressure and loss of waveform.

provide a large pulse waveform, of which the amplitude changes or loss of the signal were recorded. The third test involved placing the Finapres cuff on the index finger. After establishment of a consistent blood pressure reading, the ulnar and radial arteries were occluded one at a time, and changes in blood pressure readings or the inability to measure a pressure were noted. In all cases except the Allen's test, the second investigator was blinded as to which artery was being evaluated. This investigator recorded his findings separately. Results between the primary and second investigator were compared after completion of all the tests, which were performed on both hands of the volunteers and were completed in approximately 20 min per subject. Statistical analysis was performed using the Classification and Regression Trees (CART) analysis of Breiman *et al.* [19], or the paired *t*-test.

Results

The Allen's test was normal in all cases with the palmar blush being noted within 5 s of releasing either artery. Plethysmographic evaluation identified 49 (49/70, 70%) hands with a dominant artery. Dominance was determined when if one artery was occluded it resulted in an obviously greater decrease in the plethysmogram than occlusion of the other, nondominant artery. The ulnar artery was judged dominant in 28 (28/49, 57%) and the radial artery in 21 (43%). Since only the index finger was tested, the dominance refers only to the index finger and not necessarily the entire hand.

Evaluation of 19 hands (27%) showed indeterminate dominance, with occlusion of either artery resulting in roughly equivalent changes in the waveform. In two hands (3%) there was a complete loss of the plethysmograph waveform with occlusion of one artery, and no change in waveform with occlusion of the other artery. There was, therefore, no connection between the distal ulnar and radial arteries. Digital blood pressure findings also revealed the two cases of incomplete palmar arches, which, however, resulted in no significant change in blood pressure with occlusion of one artery, and the inability to measure any pressure with occlusion of the other artery (Fig. 1).

In 46 of 49 (94%) hands, the systolic blood pressure decreased significantly more (19.7% vs 7.0%, $p < 0.05$; paired *t*-test) with occlusion of the dominant artery than with the nondominant artery (as determined by plethysmographic waveform analysis) (Fig. 2 (a) and (b)). CART analysis [17] showed a 90% probability of arterial dominance if the systolic pressure fell more than 18%. There was also a 90% probability of the nondominant artery having been occluded when the systolic pressure fell less than 8% (Fig. 3).

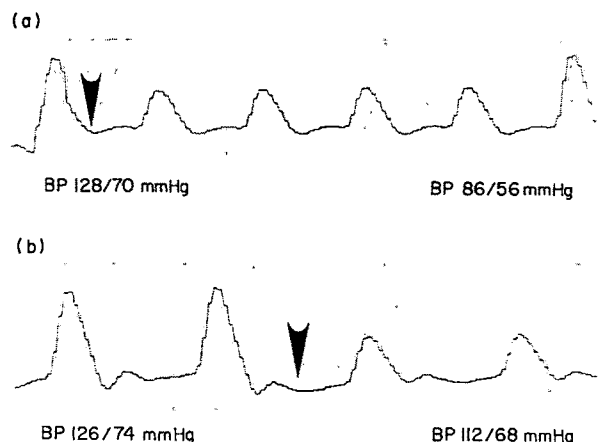


Fig. 2. (a) Digital blood pressure and plethysmograph waveform of a subject with ulnar artery dominance. (b) Same subject as 2(a). Plethysmograph waveform decreased significantly while digital blood pressure indicated the radial artery being occluded was not the dominant artery.

Discussion

The incidence of serious complications following radial artery cannulation is very low; reported as 0.2% in one study and basically nonexistent in another [20, 21]. Slogoff and Keats [20] reported a study of 1699 patients. Arterial cannulation was undertaken without any standardised methodology or techniques. Less than one-fourth of the patients were evaluated with the Allen's test. In 4% of those patients the Allen's test indicated no ulnar collateral circulation, yet none suffered serious consequences after radial artery cannulation. The authors' conclusion was that in the absence of peripheral vascular disease the Allen's test cannot be used as a predictive indicator of potential ischemia after radial artery cannulation.

Mandel and Dauchot [21] reported a study of 1000 patients undergoing radial artery cannulation. These patients were assessed with both the Allen's test and Doppler ultrasound. Only 1.8% were found to have no ulnar artery collateral circulation and radial artery cannulation was not undertaken in those patients. In spite of their fairly controlled techniques, two patients (0.2%) suffered serious complications. The authors attributed this low complication rate to adequate pre-operative evaluation and simple postoperative precautions.

Numerous articles describing refinements to the Allen's test or alternative methods of evaluation have continued to appear in the medical literature [3–9]. This would seem to indicate that clinicians are reluctant completely to espouse the findings of Slogoff and Keats. Clinicians probably realise that complications may occur. It is in the interest of patient care that obvious sources of potential complications be identified and avoided.

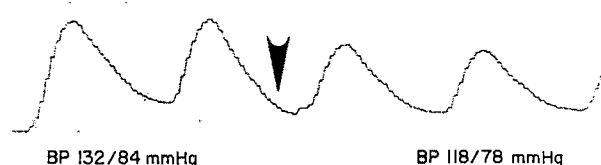


Fig. 3. Digital blood pressure and plethysmograph waveform of a subject with ulnar artery dominance shown by less than 18 mmHg drop in blood pressure with radial artery occlusion.

The introduction of electronic monitors has provided a means of evaluation that is more sensitive than the palmar blush of the Allen's test. The sensitivity of these monitors allows a standardisation of the signal, but there still exists the problem of signal interpretation. It has been noted that pulse oximeters do not always deviate in saturation readings even when flow has been severely decreased [10], while plethysmography has been shown to change in relation to flow when the monitor does not employ an auto-gain mode of waveform display [7, 8]. The display of the plethysmograph is easier to see than a palmar blush but neither is quantifiable. While not a true measurement of flow, digital blood pressure can provide a clinically relevant, quantifiable and recordable indicator of collateral circulation to the hand. Digital blood pressure should be considered when a clinician wishes to record the state of collateral circulation before arterial cannulation.

Acknowledgment

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CASE REPORT

Erythropoietin in a patient following multiple trauma

P. KRAUS AND J. LIPMAN

Summary

We report on a Jehovah's Witness who had severe blood loss following major trauma. The problems of her management without blood transfusion, and with the use of recombinant human erythropoietin therapy for severe anaemia, are described.

Key words

Complications; trauma.

Blood, haematopoiesis; erythropoietin.

Organisations; Jehovah's Witnesses.

At a time when the beneficial effects of blood transfusion are being questioned [1, 2] the admission of an exsanguinated trauma patient, who happened to be a Jehovah's Witness (JW), made us consider the reasons for improving haemoglobin concentration and the alternative means which are available to us.

Case history

A 45-year-old female Jehovah's Witness was admitted after a train accident in which she sustained multiple bilateral rib fractures resulting in a flail chest, a left haemopneumothorax, a severely contused left lung and a ruptured spleen. A splenectomy was performed at exploratory laparotomy during which time she received 3 l of crystalloid (Ringer's lactate 2 l and 5% dextrose in saline 1 l). Postoperatively she was admitted to the Intensive Care Unit (ICU), where she was found to be conscious but pale, with poor peripheral perfusion despite a blood pressure of 140/70 mmHg and a sinus tachycardia of 120 beat.min⁻¹. She had a nasotracheal tube in place and had marked surgical emphysema of the chest wall with a large anterior flail segment. She already had a left intercostal drain, and auscultation of the chest revealed distant heart and breath sounds. Her abdomen showed evidence of recent surgery and there were no other abnormalities on clinical examination.

Her electrocardiogram showed generalised nonspecific T-wave inversion with small QRS complexes. Her chest X ray confirmed the multiple rib fractures, the severely

contused, re-expanded left lung, but in addition revealed a right-sided pneumothorax which was promptly drained. Mechanical ventilation was started and her first 24 h in the unit was marked by hypotension and a decreasing urine output, for which she received fluids, initially in the form of a synthetic colloid (Haemaccel). Dobutamine (maximum dose 20 µg.kg⁻¹.min⁻¹ and dopamine (3 µg.kg⁻¹.min⁻¹) were added once it was felt that her intravascular volume had been restored. Maintenance fluid was in the form of 5% dextrose in saline. By day 7 her accumulative measured fluid balance was about 13 l positive and by day 16 it was about 23 l positive. Blood urea, electrolytes and creatinine concentrations remained normal throughout her admission. Her haemoglobin, platelets and white cell count during her time on ICU are shown in Figure 1. She was given pneumococcal vaccine on day 1 and her APACHE II score on admission was 18.

Two days after admission therapy with piperacillin and amikacin was started because of a pyrexia, increasing white cell count and worsening bilateral infiltrates on chest X ray, presumed to be the result of a nosocomial chest infection. Blood cultures and tracheal aspirates taken at that time were unhelpful.

By day 4, both of her intercostal drains had been removed and she was being fed enterally. Inotropes were withdrawn by day 9, at which stage her antibiotics were also stopped. A tracheostomy was performed on day 13. From admission her haemoglobin concentration steadily decreased to a level of 3.2 g.dl⁻¹ on day 16. This was

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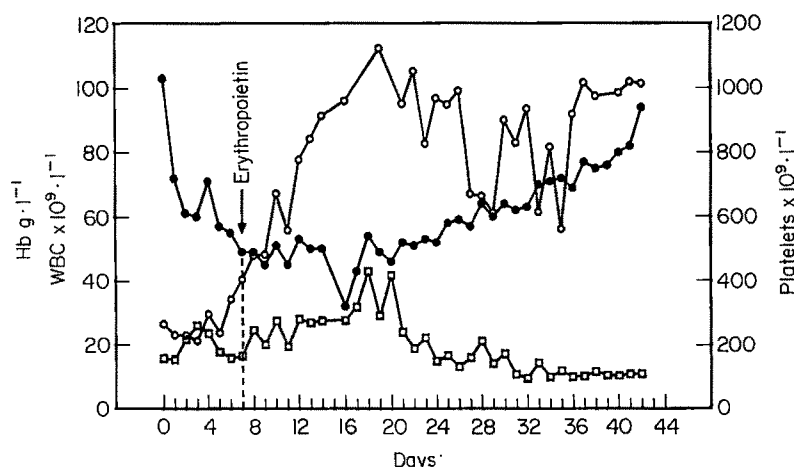


Fig. 1. Changes in haemoglobin concentration, white cell count and platelet count from admission to day 42. ●, haemoglobin concentration $\text{g} \cdot \text{l}^{-1}$; ○, platelet count $\times 10^9 \cdot \text{l}^{-1}$; □, white cell count $\times 10^9 \cdot \text{l}^{-1}$.

attributed to a combination of sepsis and continuing blood loss into the left lung which had been confirmed following re-insertion of an intercostal drain on day 14. She was given daily iron, vitamin B₁₂ and folate supplements, as well as 9 amps (4000 u each) of recombinant human erythropoietin (r-HuEPO), starting on day 7 and given over 1 week, as recommended by the manufacturers.

On day 14, ceftazidime and clindamycin were started for a new chest infection and fluid drained from her left chest grew a bacillus species which was sensitive to this antibiotic combination. She responded well and the rest of her stay was marked by a protracted period of weaning from the ventilator, after which she was eventually discharged on day 24.

She was seen again by one of us (P.K.) at the Respiratory Out-Patient Department 3 months following her accident, and she remains well.

Discussion

Under certain circumstances adequate oxygen carrying capacity can be provided by a haemoglobin of $7 \text{ g} \cdot \text{dl}^{-1}$ [1]. Although the exact critical level of haemoglobin is debatable, most intensivists would agree that this patient's haemoglobin level of $3.2 \text{ g} \cdot \text{dl}^{-1}$ needed blood transfusion. Even when her ventilatory failure, resulting from a combination of a flail chest, haemothorax, contused lung, and chest infection, was corrected it was only when her haemoglobin level began to increase that she could be successfully weaned off the ventilator. Her clinical course therefore suggests the need to transfuse a patient with such severe anaemia.

Since her religious beliefs precluded her from receipt of any blood products, her initial haemodynamic stabilisation was with fluids to achieve an adequate preload (as reflected by her central venous pressure) and intropes to maintain an adequate blood pressure and urine output. The policy of this unit is to give maintenance fluids in the form of crystalloid and to administer boluses of synthetic colloid solution as the clinical situation demands. Although we cannot exclude some element of haemodilution to explain the fall in haemoglobin, normal insensitive losses are not taken into account, nor losses into the chest and abdomen which we felt were significant. Lastly, 70% of the positive

fluid balance in the first week was accounted for in the 60 h immediately after surgery, when the patient was considered to be significantly 'behind' on fluids.

When it became obvious that these measures were insufficient to provide adequate oxygen delivery, permission was obtained for the compassionate use of r-HuEPO. This was the only option open to us since at present no synthetic blood preparations are available.

Erythropoietin has been used in patients with renal failure, haematological disorders, in other patient populations and even in Jehovah's Witnesses [3, 4]. Koestner *et al.* were the first to report its use in a Jehovah's Witness with trauma [3] and our patient is the first in whom r-HuEPO has been used to facilitate weaning from mechanical ventilation. We considered that the use of r-HuEPO was justified because the severe anaemia was one of the factors that prevented weaning from mechanical ventilation. Although her haemoglobin level would have eventually returned to normal the use of r-HuEPO was probably cost effective since it accelerated the process and shortened her ICU stay. As a result she developed only two nosocomial infections and avoided the many iatrogenic complications associated with prolonged mechanical ventilation.

Although this patient undoubtedly benefited from stimulation of her bone marrow to increase her haemoglobin concentration, the routine transfusion of critically ill patients who are anaemic is increasingly being questioned. Haemoglobin concentration is one factor in the delivery of oxygen but recent work in both adults and children [5, 6] has suggested that, although increasing the haemoglobin increases oxygen delivery, this has little effect on oxygen utilisation when compared with measures which augment the cardiac output. Although we do not suggest that this applies to levels as low as those seen in this patient, it is of interest that two recent editorials [1, 2] have questioned the level of haemoglobin at which transfusion is necessary. Furthermore, we have shown that with fluids, inotropic support and ventilatory support in which the FIO_2 never exceeded 0.6, a patient can survive, despite an acute and chronic blood loss which resulted in a haemoglobin concentration of $3.2 \text{ g} \cdot \text{dl}^{-1}$.

If transfusing patients who had previously been considered to have 'too low' a concentration of haemoglobin, is not beneficial in terms of oxygen consumption are there

any deleterious effects of giving blood? Apart from the well known complications such as transfusion reactions, metabolic and coagulation disturbances and transmission of serious diseases (e.g. human immunodeficiency virus and hepatitis B & C viruses), there is the possibility that immunosuppression promotes bacterial infection [7].

In conclusion, we have presented a patient with multiple trauma who survived without blood transfusion despite severe anaemia. The contribution of r-HuEPO to her management has been highlighted. The use of autotransfusion, r-HuEPO and synthetic blood analogues may provide a means of improving aspects of oxygen delivery and utilisation and reducing immunodepression in the critically ill in the future.

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Cimetidine–dobutamine interaction?

A. BARAKA, M. NAUPHAL AND W. ARAB

Summary

The report describes a patient during induction of anaesthesia for coronary artery by-pass grafting, in whom the infusion of dobutamine at a rate of $5 \mu\text{g.kg}^{-1}.\text{min}^{-1}$ resulted in unanticipated severe hypertension. The exaggerated response may be attributed to cimetidine–dobutamine interaction.

Key words

Inotropes; dobutamine.

Histamine; cimetidine.

Interactions; drug.

Cimetidine, an H_2 receptor antagonist, has been shown to interact with many cardiovascular drugs [1]. The following case report suggests that there may be a clinically significant interaction between cimetidine and dobutamine.

Case history

The patient, a 55-year-old man who had coronary artery disease with fair ventricular function, was scheduled for coronary artery bypass grafting. Before operation he was being treated with twice daily atenolol 25 mg. Due to peptic ulcer disease he also received cimetidine 1 g by mouth daily in divided doses.

The patient was given morphine 10 mg, promethazine 25 mg and hyoscine 0.4 mg by intramuscular injection before operation. Before and during induction of anaesthesia the electrocardiogram (ECG), arterial blood pressure (by arterial cannula) and central venous pressure were monitored. The arterial blood pressure before anaesthesia was 120/80 mmHg and the heart rate 65 beat.min^{-1} .

Anaesthesia was induced with midazolam 0.1 mg.kg^{-1} , fentanyl $50 \mu\text{g.kg}^{-1}$ and vecuronium 0.2 mg.kg^{-1} . The trachea was intubated and his lungs ventilated with 100% oxygen. Following induction, the arterial blood pressure decreased to 80/40 mmHg and an infusion of dobutamine was started at a rate of $5 \mu\text{g.kg}^{-1}.\text{min}^{-1}$. After about 2 min, the arterial blood pressure increased to 210/100 mmHg, and the heart rate to 75 beat.min^{-1} . The infusion of dobutamine was immediately discontinued to

be followed by a slow decrease to an arterial pressure of 90/50 mmHg after 15 min. Because the response did not match the expected degree and duration of action of dobutamine, it was decided to prepare a new infusion of dobutamine. However, a similar response followed initiation of the new infusion. A decrease of the infusion rate to $1 \mu\text{g.kg}^{-1}.\text{min}^{-1}$ was sufficient to maintain the blood pressure at 120/80 mmHg. A similar low infusion rate was used following uneventful weaning from cardiopulmonary bypass.

Discussion

The marked and sustained elevation of blood pressure following the short infusion of a low concentration of dobutamine before bypass suggests an exaggerated response to dobutamine.

This patient had no disease such as phaeochromocytoma or thyrotoxicosis which might explain the response and did not indulge in drug abuse with cocaine or amphetamines which could potentiate the action of sympathomimetic drugs. In contrast, the patient was prepared for operation by the selective β_1 -adrenoceptor blocker, atenolol, which can decrease rather than increase the inotropic action of dobutamine [2]. Also, chronic cardiac dysfunction can result in a selective down-regulation of the β_1 -adrenoceptors [3], which may attenuate the β_1 -adrenergic action of dobutamine on the myocardium.

Our patient had been receiving a large dose of cimetidine

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for 5 days pre-operatively, and we feel that the exaggerated response can be explained by impaired dobutamine metabolism secondary to cimetidine induced inhibition of the mixed function oxidase system of drug metabolising enzymes. Inhibition of drug metabolism by cimetidine has been found to be both dose- and time-dependent. Furthermore, cimetidine may decrease liver blood flow [4]. Both mechanisms can inhibit the overall clearance of drugs inactivated by the liver. Reduced liver blood flow will reduce the clearance of highly extracted drugs and inhibition of the oxidative enzymes will inhibit phase I biotransformation [4,5].

In contrast with noradrenaline which is rapidly inactivated by re-uptake into sympathetic nerve endings, and by the two enzymes, monoamine oxidase and catechol O-methyltransferase, dobutamine has a half-life of about 2 min, and is metabolised by the liver into inactive 3-O methyl dobutamine and glucuronide conjugates [6,7]. It is therefore possible that cimetidine may inhibit the uptake and/or the metabolism of dobutamine by the liver with a subsequent increase in the degree and duration of its action.

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The Laerdal pocket mask: effects of increasing supplementary oxygen flow

A. N. THOMAS, J. HYATT, J. L. CHEN AND S. J. BARKER

Summary

To determine the effect of increasing supplementary oxygen flow during ventilation with the Laerdal pocket mask 24 subjects ventilated a modified recording mannikin for four 90 s periods using the mask with oxygen flows of 5 l, 10 l, 15 l and 20 l.min⁻¹. Oxygen concentration increased and carbon dioxide concentration decreased with increasing oxygen flow. Tidal volume also increased with oxygen flow. The delivered oxygen concentrations were lower than previously reported at all four rates; however, mask function improved with increasing flow up to 20 l.

Key words

*Equipment; Laerdal pocket mask.
Oxygen.*

The Laerdal pocket mask (LPM) (Laerdal Medical, Stavanger, Norway) is a clear, plastic mask with a one-way valve that allows a resuscitator to ventilate a patient's lungs with expired air while avoiding possible contamination. Previous studies have shown that when used by various groups of paramedical and medical personnel, tidal volumes delivered by the mask are superior to those delivered using a bag-valve mask (BVM) system [1–3].

A modification of the LPM has added a nipple with a one-way valve to allow supplementary oxygen to be given. In the original description of this modification [4], measurements of delivered oxygen concentration were made during ventilation of one conscious subject by one resuscitator. Oxygen concentration was measured using an intermittent sampling technique, a method likely to be inaccurate when the oxygen concentration changes rapidly during a single breath. The aim of this study was more carefully to determine the effect of varying supplementary oxygen flow on the delivered oxygen concentration and delivered carbon dioxide concentration as well as tidal volume and airway pressure during artificial ventilation with the LPM. The study was carried out using a recording mannikin and subjects were recruited from within the Department of Anaesthesiology.

Methods

A standard recording mannikin (recording Resusci Annie, Laerdal Medical, Stavanger, Norway) was modified by adding two sample ports between the head and the lung. One port was connected to the sample channel of a Perkin-Elmer 1100 Medical Gas Analyzer, (Perkin-Elmer, Norwalk CT) and the other to a Honeywell 126 PC pressure transducer, (Honeywell, Pleasantville NY, USA). An Abbirico Spiroflow respirometer, (Abbirico, UK) and a Drager Series 2000 Minute Volume Meter, (North American Drager, Telford PA, USA) were added to the expiratory port of the mannikin. The mass spectrometer was calibrated according to the manufacturer's instructions at two points, using commercially produced CO₂ and O₂ mixtures; the pressure transducer was calibrated with a water column and the output signal of the respirometer was calibrated using a Hamilton one litre gas syringe (Hamilton, Whittier, CA, USA).

In order to determine the ventilatory characteristics of the mannikin the respiratory limb of a Servo 900 C ventilator (Siesmens-Elcoma, Sweden) was connected to the proximal end of the tubing normally connected to the head of the mannikin. The expiratory limb was then connected

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Table 1. Ventilatory characteristics of the mannikin during ventilation with the Servo C ventilator. Mean values and (ranges) are shown.

Delivered total volume (ml)	Breaths to 97% O ₂	Peak airway pressure PaW (cmH ₂ O)	Expired minute volume (l) (Dummy lung)	Expired minute volume (l) (Resusci Annie)
250	14.6 (14–15)	10 (10–10)	2.6 (2.6–2.6)	2.2 (2.2–2.3)
500	6.8 (6–7)	15 (15–16)	5.1 (5.0–5.1)	4.8 (4.8–4.9)
750	5.0 (5–5)	23 (23–24)	8.0 (8.0–8.1)	7.3 (7.2–7.4)
1000	3.6 (3–4)	28 (27–29)	10.6 (10.2–10.9)	9.9 (10.1–9.8)

to the expiratory port of the mannikin; the mass spectrometer sampling port was also connected to the expiratory limb.

After the mannikin had been flushed with room air the ventilator was set to deliver tidal volumes of 250, 500, 750 and 1000 ml of oxygen at a rate of 10 breath.min⁻¹ and a constant inspiratory flow and inspiratory time of 25%. The number of breaths taken for the expired oxygen concentration to reach 97%, the peak inspiratory pressure and the expired minute volume were recorded. The mannikin was flushed with room air between each reading and the whole series was repeated on five separate occasions. Results are shown in Table 1; the expired minute volume recorded by the Servo is shown both during ventilation of the mannikin and during ventilation of a dummy lung.

Twenty-four subjects were recruited from resident and faculty members of the Department of Anesthesiology. Eight of the subjects were attending anaesthetists and 16 were residents; five were female. The mean age was 33 (SD 5) years, the mean weight was 71 kg (SD 12), the mean height was 175 (SD 7) cm and the mean glove size was 7.0 (range 6–8). All subjects had previous experience with the training mannikin but not with the LPM. Subjects were given instruction in mask usage and allowed to practice with the mask for a few minutes until they were comfortable with its use.

Oxygen flow delivered to the mask was measured using two calibrated oxygen Rotameters arranged so that the resuscitator was blinded to the oxygen flow chosen. After the mannikin had been flushed with room air, the oxygen flow was set at 5, 10, 15 or 20 l.min⁻¹ and the subject was asked to ventilate the mannikin in accordance with American Heart Association standards [5] (at a volume of greater than 800 ml and a rate of 12 breath.min⁻¹) for 90 s at each oxygen flow. This time period included the time required to place the mask on the face of the mannikin, which avoided contamination with oxygen before the start of the study. Output from the mass spectrometer, respirometer, and pressure transducer was sampled at a 10 Hz cycle rate and recorded on a Personal Computer using a commercial software package (Labtech Notebook (Version 6) Laboratory Technologies Corporation, Wilmington MA, USA). The tidal volume was also recorded on the mannikin tidal volume recorder (the accuracy of which had been checked with the Servo ventilator) and the expired volume was measured with the Dräger minute volume meter. Once the expired volume exceeded 4 l the subsequent expired gas was collected in a leak-proof bag and the O₂ and CO₂ concentrations in the bag were measured at the end of each 90 s period. At the end of each of these periods the mannikin was flushed with room air and a vacuum pump was used to empty the

expired gas from the collecting bag. At least 5 min elapsed between each study time.

Oxygen flows were allocated to subjects in a random order. This was done by placing the 24 possible combinations of the four flows in individual randomly allocated plain envelopes. Each subject then received one envelope with their order of flow.

Mean delivered oxygen and carbon dioxide concentrations were calculated from the mean of all concentrations where the inflation pressure was greater than 3 cmH₂O; this included all the oxygen peaks shown graphically (Fig. 1). Peak inflation pressures and tidal volumes were measured from graphs plotted from the recorded data. The inflation pressures were divided into those less than 20 cmH₂O, between 20 and 30 cmH₂O, 30 to 40 cmH₂O, and greater than 40 cmH₂O. Tidal volumes were divided into those less than 500, between 500 and 800, and greater than 800 ml. The mean tidal volume was calculated from the division of the total volume recorded during the 90 s period with the number of breaths during that period.

Data were analysed using repeated measures analysis of variance to determine if dependent variables were significantly related to the rate of supplemental oxygen flow. If a significant relationship was demonstrated, multiple comparisons were made using the Tukey test to determine which oxygen flows differed from each other and if there was a significant linear relationship between oxygen flow and the dependent variable. Significance was set at the level of 5%.

Results

Results obtained during ventilation with the Servo C ventilator are shown in Table 1. This table shows the number of breaths taken for the concentration of oxygen leaving the mannikin to reach 97%; the table also shows peak inspiratory pressures and the returned expired minute volume expressed together with the expired volume recorded during ventilation of a dummy lung. These results show that after a delivered volume of 4 l the expired oxygen concentration is approximately equal to the delivered oxygen concentration. During use of the LPM the collecting bag was only connected to the mannikin after the expired volume had exceeded 4 l.

The peak inspired pressures generated during ventilation with the Servo ventilator are similar to those seen during ventilation of normal subjects and suggest the resistance and compliance of the mannikin would be similar to those found in a clinical setting. There was a small but persistent discrepancy between delivered and returned tidal volumes, which suggests that the expired volumes recorded in the later part of the study were approximately 10% less than

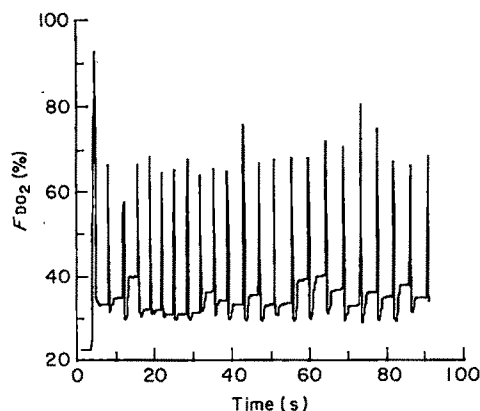


Fig. 1. Continuous recording of F_{DO_2} (delivered O_2 concentration) during manikin ventilation with the Laerdal pocket mask with 20 l supplementary oxygen flow.

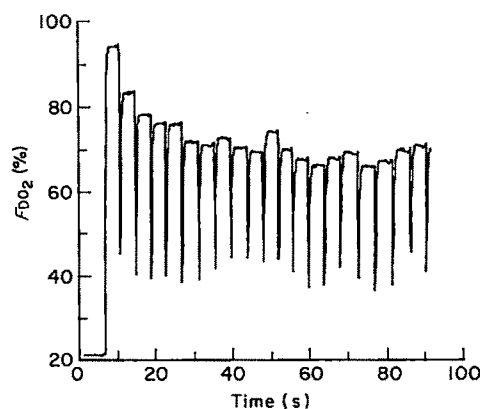


Fig. 3. Continuous recording of F_{DO_2} (delivered O_2 concentration) during manikin ventilation with the Laerdal pocket mask with 5 l supplementary oxygen flow.

the inspired volumes. There is no reason to believe that this leak should have differed over the four oxygen flows.

Figure 1 shows a typical graph of oxygen concentration recorded over a 90 s period of ventilation using an oxygen flow of 20 l.min⁻¹ delivered to the pocket mask. The brief peak oxygen concentrations represent the initial phase of inspiration, while the decrease in oxygen concentration represents dilution of the oxygen by the resuscitator's expired breath. The mean calculated delivered oxygen concentration and the mean oxygen concentrations collected in the reservoir bag at each rate of supplementary oxygen flow are shown in Figure 2. There was no significant difference between calculated delivered oxygen concentration and concentrations in the reservoir bag and both of these methods showed a significant increase in delivered oxygen concentration with increasing supplementary oxygen flow.

Figure 3 shows the typical changes in delivered CO_2 concentration during a 90 s period of ventilation with an oxygen flow of 5 l.min⁻¹ delivered to the pocket mask. The mean calculated and collected CO_2 concentrations at increasing supplementary O_2 flow are shown in Figure 4. Calculated delivered O_2 concentration was significantly higher than collected CO_2 concentration at all flows. With increasing supplementary oxygen flow, the CO_2 fell significantly in both sets of measurements.

Figure 5 shows the means of tidal volume and expired volume delivered over each 90 s period for each flow. The

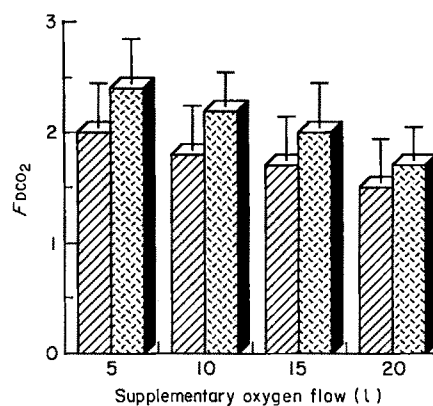


Fig. 4. Mean calculated (hatched) and collected (diagonal lines) F_{DO_2} (delivered O_2 concentration) (SD) during ventilation with the Laerdal pocket mask with increasing supplementary oxygen flow.

mean number of breaths less than 500 ml, between 500 and 800 ml, and greater than 800 ml are shown in Figure 6 together with the mean total number of breaths at each flow rate. Tidal volume and expired volume both increased significantly with increasing supplementary oxygen flow. The number of breaths greater than 800 ml increased with increasing flow and the number less than 500 ml fell.

The peak airway pressures generated during each breath

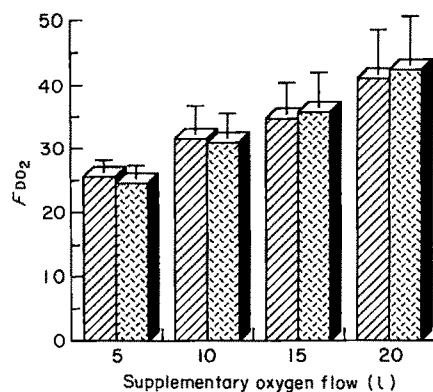


Fig. 2. Calculated (hatched) and collected (diagonal lines) mean F_{DO_2} (delivered oxygen concentration) (SD) during ventilation with the Laerdal pocket mask with increasing supplementary oxygen flow.

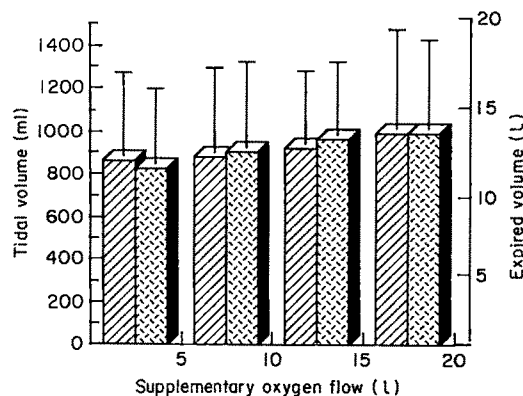


Fig. 5. Mean tidal volume (SD) (diagonal lines) and expired volume (SD) (hatched) for 90 s periods of ventilation with the Laerdal pocket mask with increasing supplementary oxygen flow.

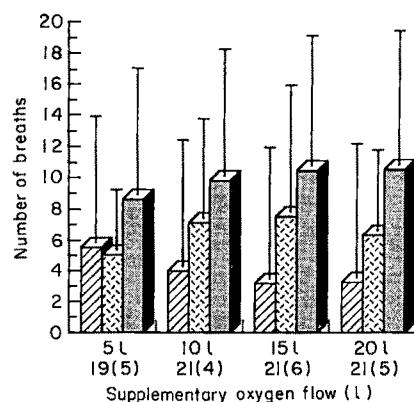


Fig. 6. Mean number of breaths (SD) of < 500 ml, 500–800 ml and > 800 ml at each supplementary oxygen flow (mean total number of breaths in each 90 s period are also shown under the X axis (SD)).

for the different supplementary oxygen flows were divided into 10 cmH₂O ranges, between 20 and 40 cmH₂O, and the number of breaths in each 10 cmH₂O range for each subject are shown in Table 2; those less than 20 cmH₂O and more than 40 cmH₂O are also shown in Figure 7. Although there was a tendency for peak airway pressure to increase with SOF, division of pressures into these ranges did not differ significantly with increasing supplementary oxygen flow. Peak inspiratory pressure exceeded 30 cmH₂O in more than 40% of breaths and exceeded 40 cmH₂O in more than 5% of breaths.

Discussion

The oxygen concentrations measured in this study are significantly less than those found in the original descrip-

Table 2. Mean number of breaths (SD) in each 10 cmH₂O range with increasing rate of supplementary oxygen flow.

PaW (CmH ₂ O)	Supplementary oxygen flow (litres)			
	5	10	15	20
< 20	2.5 (4.2)	1.6 (3.0)	1.2 (2.8)	1.2 (2.2)
20–30	9.3 (6.5)	9.8 (6.9)	9.3 (7.9)	9.1 (6.9)
30–40	6.9 (6.0)	7.6 (6.3)	8.4 (5.8)	7.5 (6.1)
> 40	0.7 (1.9)	1.5 (3.1)	2.0 (4.5)	2.4 (5.0)

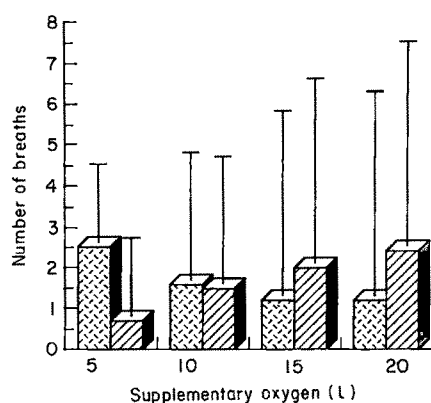


Fig. 7. Mean number of breaths (SD) when peak airway pressure was more than 40 cmH₂O or less than 20 cmH₂O for each supplementary oxygen flow.

tion of oxygen supplement into the LPM [4]. The delivered O₂ concentration is determined by the oxygen flow and the resuscitators expiratory flow, and delivered O₂ concentration will therefore change rapidly during the respiratory cycle and will vary between resuscitators and may also be influenced by the airway resistance and compliance of the patient. The larger sample size and the use of continuous oxygen measurement in our study suggests that the oxygen concentrations measured more accurately reflect true delivered O₂ concentration. Our study does, however, suffer from the limitations inherent in the use of a mannikin as an experimental model, and although we have demonstrated that the pressures generated during mechanical ventilation of the mannikin are similar to those found in clinical practice, there are other differences between patients and a mannikin model that may have some influence on oxygen concentration. In spite of this, our results show that delivered O₂ concentration during LPM ventilation is lower than expected and can only be inadequately increased by the use of high oxygen flows.

Carbon dioxide delivery during ventilation is another significant disadvantage of the LPM, and it is unfortunate that this has not been addressed in studies comparing LPM with bag–valve–mask ventilation. In this study, we have demonstrated that dilution of expired gas by supplementary oxygen reduces delivered O₂ concentration in a flow-dependent manner so that increased oxygen flow not only increases oxygen concentration but also reduces delivered CO₂ concentration. This discrepancy between calculated delivered CO₂ concentration and CO₂ concentration in the collecting bag can be explained by two mechanisms. Firstly, the expired CO₂ tends to fall during the 90 s period of ventilation and the first 4 l not collected in the reservoir bag may have a higher CO₂ concentration than that collected over the rest of the 90 s period. Secondly, the method of calculating inspired carbon dioxide does not take into account changes in flow during the inspiratory period but gives equal weight to each 0.1 s period in calculating the mean CO₂ concentration.

The mean tidal volumes exceeded American Heart Association guidelines [5] at all oxygen flows. This is an encouraging result since none of the subjects studied had had previous experience with the LPM. The significant increase in tidal volumes with increased supplementary O₂ flow is probably as a result of closure of the expiratory side of the LPM one-way valve during inspiration, so that supplemental oxygen flow is directed into the mannikin. This relationship between supplementary O₂ flow and tidal volume, which has not previously been described, represents a further advantage of increased oxygen flows.

When mask ventilation is used, it is possible for the resuscitator to generate considerable expiratory pressures, which may be useful in patients with severe obstructive or restrictive lung disease. However, in the majority of situations high peak airway pressures serve only to increase the possibility of gastric aspiration. Although the pressure required to cause gastric insufflation during cardiopulmonary resuscitation is not known, Lawes *et al.* [6] demonstrated gastric insufflation in nine anaesthetised, paralysed patients during ventilation with a Lack system, in which the expiratory valve was closed when the mean peak inflation pressure was 34 cmH₂O. However, they failed to demonstrate insufflation in a further 11 patients whose mean inflation pressure was 30 cmH₂O. Use of the Lack

system with a closed expiratory port will produce considerable levels of positive expiratory pressure and it may be that pressures required to produce insufflation with the LPM are higher than those suggested by the work of Lawes and colleagues. In spite of this, it seems likely that airway pressures of over 40 cmH₂O (found in more than 5% of the patient breaths in this study) may present a significant risk of gastric aspiration. We have been unable to demonstrate a significant relationship between supplemental oxygen flow and peak inflation pressure over 40 cmH₂O, and although such an association may well exist, the other benefits of increased SOF outweigh this potential disadvantage.

In summary, although the LPM is often used in situations where the supply of oxygen is limited, the performance of the mask is improved by increasing oxygen flow and the highest possible supplementary O₂ flows should be used up to at least 20 l.

Acknowledgments

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APPARATUS

The use of a bite raiser in the intensive care unit

P. A. WILKINSON AND G. R. WILKINSON

Summary

The use of a simple bite raiser is described to prevent self-inflicted tongue trauma in patients whose tracheas are intubated or who have tracheostomies during recovery from head injury or tetanus.

Key words

Intensive care.

Complications; trauma.

Most clinicians working in the Intensive Care Unit (ICU) are familiar with the head injured patient who traumatises the tongue and lips during the first few weeks of recovery from prolonged coma [1, 2]. Also, patients who suffer from tetanus may traumatise the lips and tongue severely as a result of the severe spasm [3] that persists for many weeks. We have used a simple bite raiser (Fig. 1) in many cases over the last 10 years and present three illustrative examples.

Apparatus

The bite raiser is simple to construct, insert, remove and clean, and allows continued mouth care. It is cosmetically acceptable to patient's relatives and inflicts no trauma to the patient during removal and insertion. The bite raiser is produced from a 3 mm sheet of soft polyvinylchloride which is vacuum moulded on to a stone model cast from an upper dental arch impression. It can be prepared in 1-2 h in an appropriately equipped dental laboratory. Similar types of device are currently in widespread use for sportsguards, temporomandibular joint disorders and dental splints following trauma [4]. The dental impression involves the use of a disposable or custom-made tray, and usually an alginate or alternatively a silicone-based impression material.

If the patient's trachea is intubated with an oral tube, it is necessary to adjust the tube to one angle of the mouth; the resulting impression registers only about 75% of the arch but this is normally adequate. A full arch impression

can be obtained from patients with a tracheostomy. In all patients, it is necessary to pack the oropharynx to facilitate the removal of excess impression material from the mouth. Increased sedation, if necessary supplemented by a muscle relaxant, may be required whilst making the impression.

Case histories

Case 1 A 4-year-old male patient suffered a fractured skull and cerebral oedema as a result of a road traffic accident. His lungs were ventilated for several days via a Jackson-Rees tube and he was noted to be chewing and biting his tongue during the weaning stage. Attempts at preventing this by McKesson props and various mouth gags were unsatisfactory due to the trauma inflicted on his small mouth. An impression was taken of the upper jaw and a simple bite raiser constructed in the orthodontic laboratory. When this was inserted, he stopped traumatising his tongue. The device was easily removed and inserted by the nursing staff for cleaning and mouth care, and was cosmetically undetectable to relatives and observers. The patient seemed to tolerate the bite raiser well.

Case 2 A 17-year-old male was admitted to the hospital with cerebral oedema and fitting after traumatic asphyxiation. He required mechanical ventilation, fluid restriction and an infusion of thiopentone. Respiratory support was required for nearly 10 days, during which time he also suffered a chest infection. During weaning from the ventilator, he was noted to be chewing and biting his tongue,

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Fig. 1. Plaster/stone model and soft vacuum-moulded bite raiser.

making mouth care very difficult. A bite raiser was constructed. The tongue healed, and no further trauma was inflicted.

Case 3 A 51-year-old male who had contracted tetanus received mechanical ventilation in ICU for 5 weeks as part of the therapy for the disease. During this time, despite huge doses of diamorphine, benzodiazepines and atracurium, he continued to traumatise his lips and tongue because of the intense spasms associated with tetanus. The nursing staff, who had cared for Case 2, requested a bite-raiser, which was used successfully and resulted in complete healing of the trauma.

Discussion

Self-inflicted trauma to the lips and tongue can be a problem in the ICU patient. Solutions suggested previously by our oral surgical colleagues have included the use of various props and gags, e.g. McKesson prop, Boyle-Davis gag and simple wooden or plastic wedges. These devices present many disadvantages, the major one being the risk of further trauma to the oral soft tissues during insertion. They are also difficult for nursing and medical staff to insert and remove, easy to dislodge, and may be totally ineffective during periods of intense spasm. Their long-term

use (more than a few hours) is not without hazard. We believe that the use of the simple bite raiser offers a safe and novel solution to the problem. Although the device requires a certain amount of technical expertise to construct, it should be within the capabilities of the Orthodontic or Oral Surgery departments of most District General Hospitals.

Appliances described by Piercell *et al.* [5] and Hanson *et al.* [2] seem to have been equally effective in similar circumstances, but are more difficult to prepare, need to be wired in place and cannot be removed for cleaning and performing oral hygiene. They fit over the mandibular rather than the maxillary arch. We suggest that the use of this simple device is a more universally acceptable solution to the problem of self-inflicted oral trauma in the ICU.

Acknowledgments

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Pre-oxygenation: the Hudson mask as an alternative technique

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Summary

The use of a simple oxygen facemask (Hudson) with high oxygen inflow (48 l.min^{-1}) was investigated as a technique for pre-oxygenation, comparing it with the Magill system (oxygen flow: $100 \text{ ml.kg}^{-1}.\text{min}^{-1}$). One hundred and thirty-eight patients scheduled for elective gynaecological and orthopaedic surgery were studied: group 1, Hudson mask and group 2, Magill system (ASA 1–2, $n = 107$); group 3, Hudson mask and group 4, Magill system (ASA 3, $n = 30$). Pre-oxygenation was assessed by measuring the times to 97%, 95% and 93% arterial desaturation (finger pulse oximetry) following 3 min of pre-oxygenation. The times taken to achieve these end-points in all the study groups suggest that the Hudson mask offers an alternative technique for pre-oxygenation.

Key words

Oxygen; delivery systems.

Equipment; breathing systems, Magill, masks, Hudson.

Pre-oxygenation is a technique widely employed in anaesthesia. Its benefits were recognised from as early as the 1950s [1–3]. There have been numerous studies of different methods of pre-oxygenation, particularly with regard to the duration of oxygen administration, which ranges from three or four vital capacity breaths to several min [4–7]. However, none of these studies have addressed a problem which is sometimes encountered in clinical practice; that of nonacceptance by the patient of a tight fitting facemask. All the current techniques studied depend on achieving a gas-tight seal with the anaesthetic facemask, which in certain patients may increase anxiety. Recently, an alternative technique using a simple oxygen mask without the gas-tight seal was investigated, using both volunteers and a lung model [8]. The purpose of the present study is to assess the efficacy of this technique in the clinical population.

Methods

Subjects and materials

The system assessed in this study consisted of a disposable oxygen facemask (Hudson Oxygen Therapy Co.) attached via a length of corrugated plastic tubing (Intersurgical)—1.5 m long, 22 mm internal diameter—to the common gas outlet of an anaesthetic machine. High flows were delivered to the mask by continuously activating

the oxygen flush device while the oxygen flowmeter was set at 8 l.min^{-1} . The total flows achieved, which were measured using a dry gas spirometer (Ohio 840), were about 48 l.min^{-1} .

After Research Ethics Committee approval, 128 adult patients scheduled for elective gynaecological and orthopaedic surgery were recruited into the study. In the first part of the study, 108 patients in ASA categories 1–2 aged less than 65 years, were randomly assigned to two groups: group 1 used the Hudson mask, and group 2 was pre-oxygenated in the conventional manner using the Magill (Mapleson A) breathing system ($100 \text{ ml.kg}^{-1}.\text{min}^{-1}$ oxygen flow). The second part of the study involved 30 ASA 3 patients who were again randomly assigned to one of the two pre-oxygenation techniques (Group 3, Hudson, Group 4, Magill). Any patient in whom it was anticipated that there might be difficulty with tracheal intubation, or risk of aspiration of gastric contents, was not studied.

Anaesthesia

In the study, patients were premedicated with papaveretum 10–20 mg and hyoscine 0.2–0.4 mg, administered intramuscularly 90 min pre-operatively. Following establishment of ECG, blood pressure and oxygen saturation monitoring, each patient underwent 3 min of pre-oxygena-

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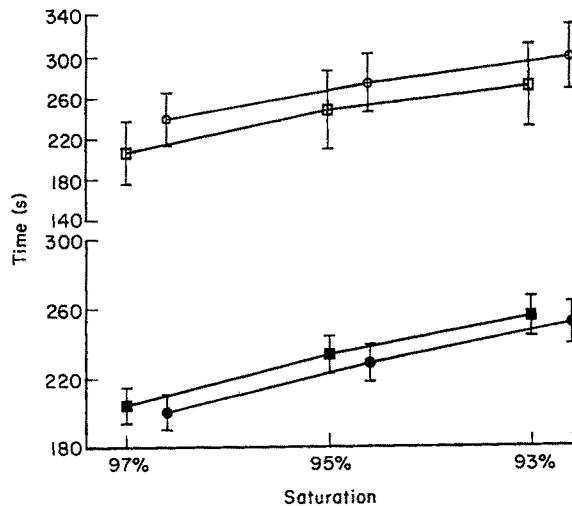


Figure 1. Graph showing the time taken to reach the arterial saturation study-points, 97%, 95% and 93%, following pre-oxygenation and induction of anaesthesia. Values are given as mean (SEM). ■ = group 1 ($n = 54$) patients in ASA categories 1–2 using the Hudson mask. ● = group 2 ($n = 53$), patients in ASA categories 1–2 using the Magill system. □ = group 3 ($n = 15$) patient in ASA group 3 with the Hudson mask. ○ = group 4 ($n = 15$) patients in ASA group 3 with the Magill system.

tion. Anaesthesia was induced with fentanyl $1 \mu\text{g.kg}^{-1}$ thiopentone $3\text{--}5 \text{ mg.kg}^{-1}$, and tracheal intubation was facilitated with suxamethonium 1.5 mg.kg^{-1} . Immediately after injection, the facemask was removed and after tracheal intubation, vecuronium 0.1 mg.kg^{-1} was administered. Ventilation was not assisted and the tracheal tube was left open to room air until the study end-point of 93% arterial oxygen saturation was reached. During the period of apnoea, anaesthesia was maintained with supplemental doses of thiopentone 25–50 mg.

Measurements

Arterial oxygen saturation (SpO_2) was measured with a pulse oximeter (Ohmeda 3700) adjusted to its fast response

mode (measurement time averaged over 3 s). The oximeter probe was applied to the thumb and the SpO_2 was recorded before and immediately after pre-oxygenation. During the apnoea period, the times for SpO_2 to decrease to 97%, 95% and 93% were measured.

Statistical analysis

Patient demographic data for the two groups in both parts of the study were compared by analysis of variance. Oxygen saturation while breathing room air and after pre-oxygenation, and the times to each desaturation point were compared using the Mann–Whitney U test. The level of significance chosen was p values of less than 0.05.

Results

The demographic data for the four groups are summarised in Table 1. In both parts of the study, there were no intergroup differences in age, weight, height, haemoglobin concentration or SpO_2 before and after pre-oxygenation. The number of men and women was similar in each group, with a similar distribution of smokers and nonsmokers. There were no significant differences in the times to 97%, 95% and 93% saturation (Table 2).

One of the patients who was assigned to the Magill system for pre-oxygenation did not tolerate breathing through the anaesthetic facemask, but when the alternative technique was offered, she did not experience any anxiety with the system. In this patient following pre-oxygenation, the times taken to reach 97%, 95% and 93% desaturation were 260 s, 337 s and 369 s respectively. Her results were excluded from the study.

No difficulty with tracheal intubation was encountered in the study and all patients recovered uneventfully.

Discussion

Anaesthetists have long acknowledged the value of administering oxygen before induction of anaesthesia. This stems

Table 1. Demographic data of patients. Values given as mean (SEM). Groups 1–2 are the patients in ASA categories 1 and 2 using the Hudson and Magill systems respectively. Groups 3–4 are the patients in ASA category 3 using the Hudson and Magill systems.

Group	Age; years	Height; cm	Weight; kg	Hb; g.dl^{-1}	Sex; m/f
Group 1 Hudson ($n = 54$)	42.5 (2.3)	165.0 (1.27)	64.0 (1.4)	12.8 (0.2)	12/42
Group 2 Magill ($n = 53$)	46.5 (2.5)	165.0 (1.27)	67.0 (1.4)	12.9 (0.2)	11/42
Group 3 Hudson ($n = 15$)	60.1 (3.4)	166.6 (3.8)	70.0 (3.2)	12.2 (0.6)	9/6
Group 4 Magill ($n = 15$)	61.7 (4.0)	170 (2.28)	74.5 (4.8)	13.2 (0.5)	7/8

Table 2. Oxygen saturation levels (SpO_2) before and after pre-oxygenation and the times to 97%, 95% and 93% desaturation. Values given as mean (SEM). Patients in ASA categories 1–2 are in groups 1–2 ($n = 54/53$) and ASA category 3 patients are in groups 3–4 ($n = 15$) $p > 0.05$.

Group	% SpO_2 breathing air	% SpO_2 after pre-oxygenation	Time to 97% SpO_2 in s	Time to 95% SpO_2 in s	Time to 93% SpO_2 in s
Group 1 Hudson mask ($n = 54$)	96.9 (0.3)	99.8 (0.1)	204.6 (10.7)	231.6 (10.7)	253.1 (11.6)
Group 2 Magill ($n = 53$)	96.2 (0.3)	99.8 (0.1)	200.7 (10.2)	228.9 (10.5)	251.4 (12.1)
Group 3 Hudson ($n = 15$)	96.0 (0.8)	99.5 (0.2)	206.4 (30.9)	246.2 (38.4)	270.9 (39.9)
Group 4 Magill ($n = 15$)	95.1 (0.8)	99.4 (0.1)	239.6 (26.1)	274.2 (28.3)	298.7 (30.7)

from the analysis of arterial samples taken in patients during the period of apnoea [9–10]. In the last few years the ubiquitous adoption of pulse oximeter monitoring has reinforced the benefits of this routine in anaesthetic practice. Kung and colleagues [11] undertook a controlled study of SpO_2 using pulse oximetry in healthy patients during induction of anaesthesia and they concluded that some form of pre-oxygenation should be used in all patients receiving general anaesthesia. However, they, like many anaesthetists, recognise that patient acceptance is the major obstacle to routine pre-oxygenation. Some patients find the application of a tight fitting facemask distressing and in the case of facial injuries or burns it is difficult to attain an air-tight seal.

In the present study, a few of the patients in the Magill system group were apprehensive of breathing through the tight-fitting face-mask and one patient refused to cooperate, despite repeated reassurances. When offered the Hudson mask technique, she underwent pre-oxygenation without any difficulty. No patients in the Hudson mask group expressed any concern or misgivings about the technique.

Various methods have been described to improve patient tolerance including the application of nose clips and breathing with lips pursed, through a tracheal tube connected to the breathing system [12]. Such options, however, still depend on a cooperative patient. The technique described in this study offers a simple, readily available solution to the frightened patient. The functional characteristics of oxygen treatment masks such as the Hudson have been elucidated previously [13]. To ensure an inspired oxygen concentration of 100%, the oxygen inflow into the mask must equal or exceed the peak inspiratory flow (PIF) of the patient. Since the PIF of adults during quiet breathing varies between 20 and 35 l.min⁻¹ [8, 14, 15], an oxygen inflow rate of 48 l.min⁻¹ will exceed the PIF of most resting adults.

This study demonstrates that there is an effective addition to the current techniques of pre-oxygenation in patients in ASA categories 1–2. The results also suggest that the Hudson mask with high flows offers a suitable alternative for pre-oxygenation in patients in ASA category 3. In these patients, there were intergroup differences in the mean times to the SpO_2 points, but these did not achieve statistical significance. Additionally, it is also interesting to note that there were differences in the times between patients in ASA categories 1 and 2 and 3 particularly the time to reach 93% saturation. This may be explained by the inherent differences in uptake of oxygen, due to cardiac output changes or oxygen consumption, in these groups, of patients. The lack of statistical significance in this instance may reflect the power of the study since the sample size in

this heterogeneous group of patients in ASA 3 category may not have been sufficient to detect any difference in the times to attain the various saturation end-points. Future clinical studies are needed therefore before the technique can be recommended in this group of patients.

In conclusion, the values obtained for the time taken to reach the SpO_2 points in this study suggest that pre-oxygenation using the Hudson mask with high oxygen inflow, is comparable to the standard technique of using the Mapleson A system. However, the authors recommend caution before extrapolating these findings to patients who present for emergency surgery, particularly in those who are apprehensive and in pain, in whom peak inspiratory flow rates may be excessive.

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The early development of pain relief clinics in the UK

M. SWERDLOW

Specific treatment of pain first became feasible in 1884 following the demonstration by Karl Koller [1] of the local anaesthetic properties of cocaine. This discovery rapidly led to the use of cocaine injections for the relief of trigeminal neuralgia and many other types of chronic pain. However, the relief was, of course, temporary and more worthwhile results only became possible in 1902 when Schlosser [2] successfully used alcohol nerve blocks to treat patients with severe trigeminal neuralgia. Within a few years, alcohol block of many other nerves was being tried for a variety of other pain conditions including those involving the sympathetic nervous system. A further advance was made in 1931 when Dogliotti [3] introduced the technique of intrathecal alcohol injection. A report of 18 patients with cancer pain who were successfully treated in the UK by this technique was published by Dr Richie Russell [4] in 1936.

All this pioneer work was carried out by surgeons, mostly in Germany and the USA, and by the early 1920s various types of nerve block were being routinely used for pain relief by surgeons in many countries. By this time however, the range of possible surgery was expanding and surgeons' interest in nerve blocks diminished. Nerve blocking for pain syndromes therefore gradually came into the province of anaesthetists who were already using local anaesthetic blocks to provide anaesthesia for surgery. This explains why the first clinic ever to be established to receive only patients suffering from chronic pain was started by an anaesthetist, Dr E. Rovenstine, at Belle Vue Hospital, New York. Over the course of the next few years, a number of such nerve block clinics were started in the United States.

Largely because of the advent of the War, the idea did not spread quickly to other parts of the world. In particular, it was not until 1947* that the first (nerve block) clinic was opened in Great Britain. This was organised by Dr B.G.B. Lucas at University College Hospital, London. Shortly after the War, Lucas spent one month at the Mayo Clinic (personal communication) where he learned pain therapy nerve blocks from John Lundy. Lucas was interested in facial and dental pain and at first he performed nerve blocks for facial pain in association with radiotherapists and oral surgeons in accommodation provided by the radiotherapy department. Later, he was involved in general pain management.

In the following year (1948), Dr Eric Angel opened a

*Pain clinics did not appear in mainland Europe until 1960 [5].

clinic in Plymouth. Eric Angel, while in the RAF, was seconded on several occasions to the Nuffield Department in Oxford where he saw nerve blocks being performed by W.W. Mushin. In the RAF he also met Bernard Lucas and to quote Angel (personal communication) 'In about 1947 I was lunching with him (Lucas) at UCH, and he said "I've got to dash down to my Pain Clinic", which remark intrigued me and then after talking to him about it I decided to try and get one in Plymouth. It was difficult to get the surgeons to agree but I finally managed ... it was designated the Pain and Diagnostic Clinic'. Angel also says that some little time later he was in Liverpool with Dr Cecil Gray who was anaesthetising for Professor Charles Wells. 'I was telling Cecil Gray about the clinic and Charles Wells said "I think we ought to have one of those Cecil".' Shortly afterwards a clinic was started at the Liverpool University Hospital; this was run by Cecil Gray until 1949 and after that by Dr E. Riding. A report on more than 5 years' experience in the use of drugs in this clinic was published by Dr J.W. Dundee in 1957. Dundee [6] reports that in the first year of the Liverpool clinic 48% of patients were treated by nerve block alone and 52% had analgesic drugs in addition to nerve block. By 1956 about 40% received drugs alone, 28% were treated with drugs plus nerve block and 32% were given a nerve block alone.

On 7 January 1949 Dr Ronald Woolmer [7] gave a paper to the Section of Anaesthetics of the Royal Society of Medicine (RSM) on therapeutic and diagnostic applications of anaesthesia. The content mostly related to the use of autonomic nerve blocks for pain of autonomic nervous aetiology, but it also dealt with chemical neurolysis for cancer pain and nerve blocks for postherpetic neuralgia and for sciatica. At the end of the paper appears the sentence 'it is encouraging to find that nerve block clinics, attached to Departments of Anaesthesia ... are beginning to find a place in this country'. One of the other earliest British papers on pain treatment was by Douglas Wilson [8] of Londonderry. This deals mainly with autonomic nerve block, particularly in relation to Raynaud's and Hirschprung's diseases, but it also includes the use of intrathecal alcohol for cancer pain. Wilson makes particular mention of 'intravenous analgesic therapy', recommending for this purpose a mixture of 7.5% alcohol, 5% procaine and 5% dextrose. He considered that the technique 'may get great attention in the future as the only

complication encountered is inebriation due to rapid administration'.

Another pioneer was Dr J.R.J. Beddard, who, in 1942 attended a demonstration of nerve blocks (for surgery) by N. R. James at the West Middlesex Hospital. In 1943 Beddard was posted to a Military Hospital in West Africa where he performed over 500 nerve blocks to provide surgical anaesthesia. On returning home to the Royal United Hospital, Bath, he used this form of anaesthesia and since 1951, when he carried out his first chemical neurolysis (for trigeminal neuralgia), he treated 80 patients with cancer pain by permanent nerve blocks [9].

Dr H.C.J. Ball introduced the concept of a pain relief clinic, at first on an *ad hoc* basis, at Southampton General Hospital in 1954. Ball and his colleagues [10] later reported 411 therapeutic nerve blocks carried out between October 1954 and June 1963. In 1955 Belam and Dobney, respectively an anaesthetist and a specialist in physical medicine, were managing a joint pain clinic at the Whittington Hospital. They published a report [11] in 1957 of their first 100 cases treated by nerve block.

Whilst at that time the attention of anaesthetists was mostly devoted to the use of nerve blocks and, to a lesser extent, of analgesic drugs, neurologists and neurosurgeons were considering specific pain syndromes and various means of dealing with them. Thus at a symposium on intractable pain at the neurology and psychiatry section of the BMA Annual Meeting in 1949 [12], Wylie McKissock reported on experience with division of the spinothalamic tract and with bilateral prefrontal leucotomy for relief of pain. Richie Russell described his method of repeated percussion of stump neuromata in the treatment of painful amputation stump and phantom limb, and Russell Brain showed a patient in whom severe thalamic hypersensitivity to local stimulation was suppressed by a stimulus to the same spot on the unaffected side of the body. Another paper by a neurologist (Dr J. Penman) also antedates more recent pain clinic 'discoveries'. Penman reports [13] on 275 patients who were relieved of *tic douloureux* by trigeminal alcohol injection. The conclusions at the end of his paper are well worth quoting: 'Doctors who relieve such pain should study the personalities of their patients before treatment, try to pick out those who are attached to their pain and be ready to deal themselves with subsequent emotional disturbance. Prevention by relief of the pain before it can become chronic is of course the best way'. This last sentence anticipated plasticity by some 30 years! Another nonanaesthetist who must be mentioned is Robert Maher of Rochdale who, among other things, introduced the technique of intrathecal phenol injection for cancer pain [14].

In the 1950s the pain relief clinic continued to have as its main function, the administration of nerve blocks. In fact, during a research Fellowship in Pittsburgh, Pennsylvania in 1954, I remember spending a great deal of time in the pain clinic assisting with nerve blocks, both diagnostic and therapeutic, although we were then starting a number of pharmacological studies. On returning home I started, on an *ad hoc* basis, to see patients referred by hospital colleagues and in 1959 at the suggestion of the senior surgeon I opened a regular (at first one session a week) pain relief clinic. A preliminary report was published in 1963 [15].

Over the next 10 years, a number of other pain relief clinics appeared. The usual pattern was that an anaesthetist

would start doing nerve blocks for chronic pain at the request of hospital colleagues. The block(s) would be carried out in an anaesthetic room or in an anteroom of the operating theatre suite either before or after the operating list or even *in lieu* of the anaesthetist's lunch. As the value of the service proved itself and the number of patients referred for treatment increased, a half day or full day would be set aside for this work and the service would be moved to the hospital outpatients department as a regular clinic. The geographical location of pain relief clinics in the country at that time depended more on the enterprise and enthusiasm of the individuals initiating them than on the reputation of the hospitals involved.

Two clinics which were opened at this time deserve special mention because they were initially multidisciplinary. The clinic at Bristol was started in about 1963 and was conducted by an anaesthetist (John Challenger) a psychiatrist and a neurologist. Curiously the psychiatrist and neurologist decided after a while that there was no advantage in joint consultations and these were discontinued. Richie Russell established a Pain Relief Clinic at the Churchill Hospital, Oxford in 1964. This was organised by a neurologist, a psychiatrist and an anaesthetist (Dr John Lloyd) but the work was gradually left to the anaesthetist (personal communication).

By the end of the 1950s, it had become clear that a diagnosis of the cause of pain was an essential precursor to the initiation of treatment; we also needed to know a great deal more about the nature of pain and of patients with chronic pain. Vandam and Eckenhooff [16], writing of their experience in the (nerve block) clinic in Philadelphia, gave the following graphic and in some ways prophetic description of their opinion of nerve block therapy:

'Success was achieved primarily in those patients suffering from acute somatic pain or pain associated with autonomic nervous system imbalance. We never believed that all pain would respond (to nerve block) but neither were we prepared for the number of total failures or the high incidence of insignificant improvement. Perhaps the time spent in what seemed to be a fruitless endeavour might have been better expended in studying the basic nature of the problems encountered. The answer may lie not so much in the development of new techniques of injection or synthesis of new agents as in an integrated approach to the unknown features of pain'. They recommended the formation of multidisciplinary pain units, this several years before Bonica started his efforts to achieve this end.

Anaesthetic textbooks were an important source of learning in the 1950s, but there was still no mention of pain clinics in the books of that decade. The first textbook to treat pain relief as a separate subject was the first edition of *A Practice of Anaesthesia* (Wylie and Churchill-Davidson, 1960). The 1953 and 1959 editions of *Synopsis of Anaesthesia* (J.A. Lee) each had two pages on the use of somatic and autonomic blocks to relieve pain but it was not until later editions that the subject of pain clinics was included.

In the mid 1960s pain relief clinics were still few and far between. I certainly felt (despite 'booster doses' of pain clinic experience in the USA in 1957 and 1959) that meeting others engaging in this sphere of work would be mutually beneficial. Accordingly, in the Spring of 1967 I wrote to every pain relief colleague that I could discover in the UK (about 29 in all) inviting them to a meeting to

exchange ideas and experiences. The response was very encouraging.

The initial meeting was held at the Postgraduate Medical Institute of Salford University on 10 November 1967. The 17 'founder members' who attended were: Dr J. Anderson, Edgeware; Dr A.S. Brown, Edinburgh; Dr J.M. Burn, Southampton; Dr J.H. Challenger, Bath; Dr Anne Laughland, Glasgow; Dr R.M. Maher, Rochdale; Dr M. Mehta, Norwich; Dr H. Raftery, Dublin; Dr R.N. Sinclair, Glasgow; Dr G.C. Steele, London; Dr J.H. Stevens, Cambridge; Dr M. Swerdlow, Manchester; Dr E. Thomas, Birmingham; Dr T.C. Thorne, Rochford; Dr J.A. Thornton, Sheffield; Dr R.P. Wise, London; Dr M.D. Vickers, Birmingham.

It will be realised that all except Dr Maher were anaesthetists. Letters of apology were received from Professor Mushin and Drs Bryce-Smith, Riding, Angel and Belam.

My chairman's welcoming speech at the beginning of the Meeting included the following: 'I feel that many of us are working to some extent in isolation and getting together and exchanging experiences, techniques and complications can only do each and all of us a lot of good. Personally I often feel that my results are quite inadequate and I would like to know how one could more effectively relieve what is very obvious suffering. I am sure that this meeting will provide some of the answers. This meeting, as I see it, is largely an exploratory one and I have not arranged any formal papers; I thought rather that we could spend the time in open discussion of the problems from various aspects. However, in order that the discussion should not be too diffuse, I thought that we ought to have some sort of boundaries and I suggest we divide the day into four sections: (1) the organization of a Pain Relief Clinic; (2) pain patients, pain conditions and pain problems; (3) nerve blocks and other methods of treatment; (4) evaluation of results and complications.'

The session on 'other methods of treatment' in fact turned out to be about opioids, chlorpromazine and a brief mention of cordotomy.

There was lively discussion throughout the day and at the conclusion it was unanimously agreed that in future similar meetings should be held each year.

The second meeting of the embryo International Pain Society (I.P.S.) took place in Cambridge in October 1968 with Dr J.H. Stevens as host. Once again the one-day meeting took the form of a general discussion without formal presentations.

In the following year Dr J.H. Challenger arranged the Meeting in Bristol. In addition to general discussion, there were papers by individual members on 'the neurolytic effect of phenol', 'the collection of information' 'phantom limb pain' and 'the multidisciplinary approach to pain relief'. An attempt was made at this meeting to initiate a combined study of the results of pain relief treatment in patients with (a) cancer, (b) post-herpetic neuralgia, (c) phantom limb pain. A questionnaire was compiled and circulated to members for completion. In fact only 100 completed forms were returned, 80 of which related to cancer pain but these provided some useful information.

The 1970 meeting was organised by Dr D. S. Robbie at the Royal Marsden Hospital, London. At this meeting the main subject of general discussion was notekeeping. Papers were given by members on 'The relation between pain and personality in patients with advanced cancer' 'thermo-

graphy in the diagnosis of sympathetic dystrophy' and 'experiences with percutaneous cordotomy'.

The Pain Group was now firmly established and steadily growing; its activities were being regularly reported in *Anaesthesia*. However, the pattern of its activities, although attractive and enjoyable, was giving the organisers some food for thought. It was felt that the lack of structure of the meetings tended to result in repetitiveness of discussion and that perhaps the value of the meetings might be enhanced by having invited speakers on topics of special interest. It was also considered that too much time was spent discussing administrative matters and many members thought that some definition of criteria for membership should be introduced. The time had clearly come to formalise the organization by appointing a small executive.

At the 1971 annual meeting in Birmingham with Dr Eugene Thomas as host, the members decided to constitute the group into 'The Intractable Pain Society of Great Britain and Ireland' and elected the following to a 3-year term of office: Chairman, Dr M. Swerdlow; Hon. Treasurer, Dr J.H. Challenger; Hon. Secretary, Dr M. D. Churcher.

It was decided that membership should be restricted to medical practitioners actively engaged in the treatment of patients with intractable pain. It was agreed that there should be an annual subscription of £1 to cover expenses.

Post script In 1977, 10 years after the formation of IPS, a questionnaire was sent out to all members; 59 replies were received. Twenty-nine reported that their clinic had existed for longer than 5 years, and 17 were of 1–5 years duration. Forty-three respondents had officially recognised sessions, 18 of whom had two or more pain sessions per week. One finding, which contrasted happily with the state of affairs when the Society was founded, was that 'only three members ever work in isolation away from other consultant support'. Incidentally, the membership list for 1977 showed that only eight of the 17 founder members were still engaged in the practice of pain relief.

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Forum

Influence on hearing of 22 G Whitacre and 22 G Quincke needles

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Summary

Audiograms were performed pre-operatively and 2 days postoperatively in 48 patients given spinal anaesthesia for transurethral resection of the prostate. Hearing levels were examined at 1000 Hz and below. Either 22 G standard design (Quincke) needles ($n = 25$) or 22 G pencil-point design (Whitacre) needles ($n = 23$) were used. Hearing loss of 10 dB or more at two or more frequencies were observed in six of 25 patients in the Quincke group and in two of 23 patients in the Whitacre group. The mean hearing level was more reduced in the Quincke group. The shape of the tip of the spinal needle seems to be of some importance to the effects on hearing level that may occur after spinal anaesthesia.

Key words

*Anaesthetic techniques regional; spinal.
Ear, hearing; loss.*

Recently major hearing losses have been reported after spinal anaesthesia [1–3] and minor hearing deficits have also been demonstrated [4, 5]. The incidence of the latter has been correlated to the diameter of the spinal needle used for the lumbar puncture and such minor hearing deficits may occur without the concurrent presence of a postspinal headache, indicating that audiometry may be a sensitive indication of cerebrospinal fluid leakage [5]. Among several factors, the incidence of postspinal headache is related to the shape of the spinal needle tip [6] and recently an *in vitro* investigation has shown that dural leakage is also related to the shape of the spinal needle tip [7].

This study aimed to determine the incidence and the degree of vestibulocochlear dysfunction in patients who underwent transurethral resection of the prostate (TURP) under spinal anaesthesia performed using either 22 gauge (G) standard design (Quincke) needles or 22 G pencil-point design (Whitacre) needles, (Fig. 1) to determine whether the shape of the needle tip is of importance. Any possible correlation between hearing loss and postspinal headache was also investigated.

Method

After Ethics Committee approval, informed consent was obtained from 48 patients scheduled for TURP under spinal anaesthesia. Patients suffering from systemic disorders apart from arteriosclerosis, a history of deafness, inspissated cerumen and an inability to cooperate during audiometry were not studied.

The audiological evaluation consisted of pure tone audiometry at the frequencies of 125, 250, 500 and 1000 Hz using the ascending technique and was performed by experienced audiometrists. A Madsen OB 70 or OB 522 audiometer (Madsen Electronics, Herlev, Denmark) was

used and the system calibrated according to the international standard (ISO 389, 1975). Audiometry was performed in double-walled silent chambers. The patients were randomised to receive spinal anaesthesia using either a 22 G Quincke or a 22 G Whitacre needle (Braun Melsungen, Germany). On the morning of operation an audiogram was performed in each patient. Premedication was with pethidine 25 mg and dixyrazin 10 mg (a phenothiazine derivative) given intramuscularly. After intravenous infusion of at least 500 ml of compound sodium lactate solution, spinal anaesthesia was administered in the L₃₋₄ interspace using 3.5 ml of 0.5% bupivacaine in 8% glucose. One dural puncture was allowed in each patient; if an additional dural puncture was necessary the patient was removed from the study. The Quincke needles were inserted with the bevel parallel to the longitudinal axis of the spinal cord. Systolic arterial blood pressure was measured by sphygmomanometry at 5 min intervals during the operation, and any untoward event was recorded. The level of analgesia was estimated by the pin-prick method after 20 min. All patients remained recumbent for the first 12 h after surgery. Per- and post-operatively, fluids were given in order to keep the haematocrit at 30%. On the second postoperative day another audiogram was performed in each patient under the same conditions as the pre-operative examination. The audiometrist and the patient were unaware of the type of needle used. Each patient was also examined at that time by an anaesthetist to determine the presence of a postspinal headache or dysfunction of the third, fourth, sixth, seventh or eighth cranial nerves.

Statistical methods

All results are presented as mean (SEM). In each pair of audiograms from one patient the change of hearing level

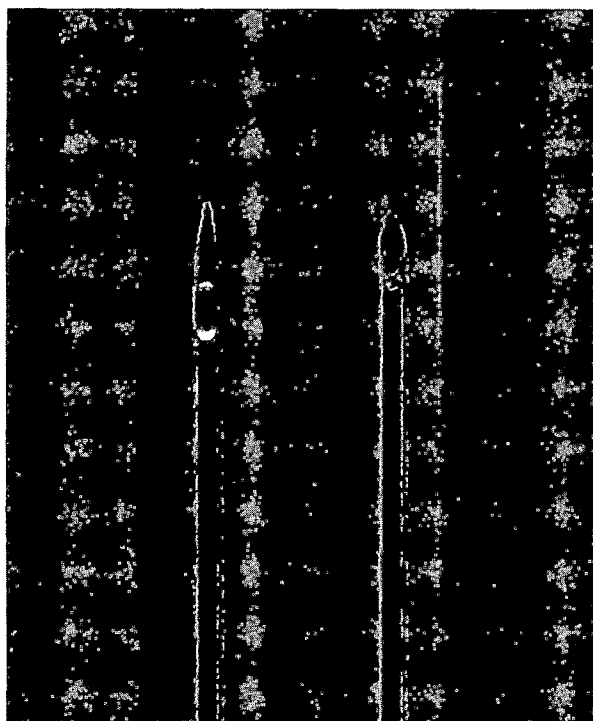


Fig. 1. The point of a Whitacre needle (left) and of a Quincke needle (right).

was calculated at each frequency as was the mean change of hearing level for right and left ears respectively for all patients in each of the two groups. A 'worse side' was defined as the side on which hearing loss was greater and the results were analysed for statistical significance using a *t*-test. Student's *t*-test for independent samples was used to establish the possible statistical significance of demographic and audiometric differences between the groups. Values of $p < 0.05$ were accepted as indicative of statistical significance.

Results

Age, weight, and height were similar in the two groups, as was the analgesic level (Table 1). The mean maximum decrease in systolic arterial blood pressure was statistically significant and similar in both groups. No patient had symptoms or laboratory evidence of the 'TUR-syndrome' [8] and there was no difference between the groups in postoperative haematocrit or sodium concentration. One patient in the 22 G Quincke group complained of unilateral tinnitus of 4 h duration on the first postoperative

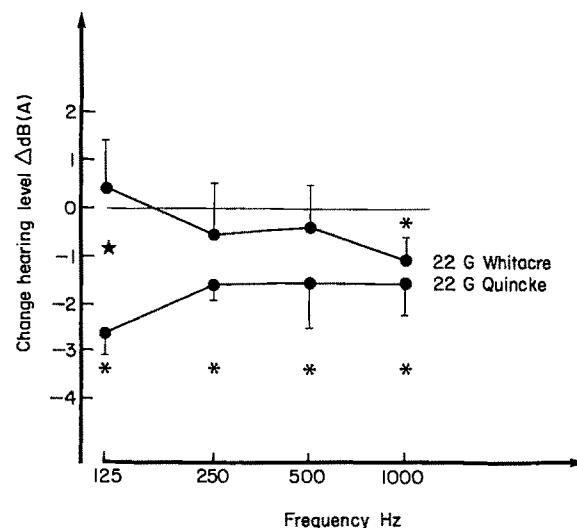


Fig. 2. Mean change in hearing level (SEM) on 'worse side' is shown at each frequency. Negative values indicate decreased hearing. *, Statistically significant change in hearing level compared to pre-operative values; ★, Statistically significant intergroup difference.

day; the symptom was unaccompanied by headache. In the 22 G Whitacre group, two patients complained of nonpositional headache of short duration on the first postoperative day, and one patient complained of nausea during the second postoperative night. Neither of these patients revealed audiometric signs of vestibulocochlear disturbances. Apart from the audiometric changes, no postoperative cranial nerve palsies were detected in either group.

Audiometric results

In the 22 G Quincke group (Table 2 and Fig. 2) there was a statistically significant and unilateral hearing loss at all frequencies and in six of the patients (24%) there was a decrease in hearing level of 10 dB or more at two or more frequencies. In the 22 G Whitacre group (Table 2 and Fig. 2) a statistically significant and unilateral hearing loss was only observed at 1000 Hz and two of 23 patients (9%) revealed hearing losses of 10 dB or more at two or more frequencies. There was a statistically significant intergroup difference in the reduction of hearing level at 125 Hz where hearing loss was greater in the 22 G Quincke group.

Discussion

The previously reported incidence of vestibulocochlear dysfunction after spinal anaesthesia varies between 0.2% and 8% [3, 9], based on the incidence with which patients complained of deafness (i.e. a major hearing deficit). However, minor hearing deficits after spinal anaesthesia have been demonstrated on the second postoperative day in 42% of patients who underwent TURP [4]. A recent study [5] indicates that hearing deficits are related to the diameter of the needle used for the lumbar puncture and the same study suggests that the dural tear caused by the spinal needle may lead to a fall in the cerebrospinal fluid pressure.

In most previously published reports of hearing loss after spinal anaesthesia, the hearing loss occurs within the low-frequency range [1–5], that is between 125 and 1000 Hz; the hearing level was therefore only examined at frequencies below 1000 Hz in this study. The mean hearing deficits

Table 1. Patients' demographic details expressed as mean (SEM).

	22 G Quincke needle (n = 25)	22 G Whitacre needle (n = 23)
Age; years	68.1 (1.5)	66.9 (1.5)
Height; cm	174 (1.2)	173 (1.1)
Weight; kg	77.5 (2.3)	75.3 (1.4)
Level of analgesia; thoracic segments	6.7 (0.4)	6.2 (0.3)
Pre-operative systolic arterial blood pressure; mmHg	152 (4)	153 (7)
Maximum decrease in systolic arterial blood pressure; mmHg	43 (4)	48 (4)

Table 2. Change in hearing level (dB) between pre- and postoperative values. Results are expressed as mean (SEM). Negative values indicate hearing loss.

	22 G Quincke needle (n = 25)		22 G Whitacre needle (n = 23)	
	Right ear	Left ear	Right ear	Left ear
Frequency; Hz 125	-1.4 (1.0)	-2.6 (0.5)*	0.4 (1.0)	1.1 (1.4)
250	-1.6 (0.3)*	-1.2 (1.1)	1.3 (1.0)	-0.6 (1.1)
500	-1.6 (0.9)*	-1.2 (1.0)	0.2 (0.7)	-0.4 (0.9)
1000	-1.4 (0.8)	-1.6 (0.6)*	-0.4 (0.5)	-1.1 (0.5)*

*p < 0.05.

demonstrated in this study are small and of no clinical importance. Hearing losses after spinal anaesthesia are usually transient, although they may take several months to disappear [1-4, 9-10].

The study by Hart and Whitacre [6] demonstrated a 50% reduction in the incidence of postspinal headache by using a pencil-point spinal needle compared to the incidence seen when a Quincke needle was employed. The same study suggested that a noncutting (Whitacre) needle would cause less damage to the dural fibres and thus may reduce the cerebrospinal fluid leak after lumbar puncture. A more recent study has demonstrated this *in vitro* [7]. Also, a low incidence of postspinal headache (1.8%) has been demonstrated when spinal anaesthesia was performed using a 22 G Whitacre needle [11] whereas the incidence of postspinal headache using a 22 G Quincke needle varies between 9 and 36% [10, 12]. In this study the use of a Quincke needle resulted in small but statistically significant unilateral reductions in hearing level at 125, 250, 500 and 1000 Hz, whereas the use of a Whitacre needle only resulted in a unilateral reduction of hearing level at 1000 Hz (Fig. 1).

The aetiology of vestibulocochlear dysfunction after spinal anaesthesia remains unknown. It is possible that the decrease in cerebrospinal fluid pressure that follows dural puncture is a result of leakage through the dural tear caused by the spinal needle. The decreased pressure may be transmitted via the cochlear aqueduct to the perilymph in the inner ear causing an endolymphatic hydrops. Endolymphatic hydrops is involved in the pathogenesis of Meniere's disease [13, 14] and the initial hearing loss observed in these patients also occurs within the low-frequency range and the hearing loss may be unilateral [13, 14]. Changes in cerebrospinal fluid pressure have been shown to induce changes in the perilymphatic pressure in the inner ear [15]. Thus, the differences in cerebrospinal fluid leakage is the most likely explanation for the audiometric results in this study. The present study was limited to the first two postoperative days because of the early discharge of patients from this hospital, so although the majority of cases of postspinal headache occur within 2 days of spinal anaesthesia [1, 9, 10, 16], it is possible that some potential cases of postspinal headache were missed.

The two groups were similar in the degree of systolic hypotension, the dose of bupivacaine used, age, weight, height and level of sensory blockade. Thus the most likely explanation for the observed hearing changes is that differences in the designs of the needles caused differences in the dural tears allowing different amounts of cerebrospinal fluid leakage to alter the cerebrospinal fluid pressure. The study suggests that the shape of the tip of the needle used

for lumbar puncture is of some importance to the change in hearing level that may occur after spinal anaesthesia.

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Baroreflex control of heart rate during high thoracic epidural anaesthesia

A randomised clinical trial on anaesthetised humans

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Summary

Baroreflex control of heart rate after cardiac sympathectomy induced by thoracic epidural anaesthesia was evaluated in 30 patients who were randomly assigned to group 1 (bupivacaine 0.25%), group 2 (bupivacaine 0.5%) or group 3 (control). Plasma volume expanders were given to equalise preload conditions, as assessed using transoesophageal echocardiography. All measurements were made under general anaesthesia. Baroreflex sensitivity was determined from the heart rate response to phenylephrine and nitroglycerine. There was no difference in cardiac slowing in response to phenylephrine between the three groups. Baroreflex sensitivity, measured as cardiac acceleration in response to nitroglycerine, was significantly lower ($p < 0.01$) in groups 1 and 2 (1.8 and 1.5 ms.mmHg^{-1} respectively) compared with group 3 (3.5 ms.mmHg^{-1}) with no differences between the two bupivacaine concentrations. The results suggest that baroreflex-mediated response to decreases in arterial pressure is dependent on the integrity of the sympathetic nervous system.

Key words

Anaesthetic techniques regional; epidural.

Reflexes; baroreceptor.

Heart; cardiovascular reflexes.

Sympathetic nervous system; blockade.

It has been suggested that cardiac sympathectomy induced by high spinal or epidural anaesthesia may alter arterial baroreceptor reflexes [1–3]. At present, little information is available about the extent and clinical significance of such alterations. One study by Takeshima and Dohi surprisingly showed that cardiac sympathectomy induced by cervical epidural blockade did not alter heart rate response to decreasing blood pressure, but suppressed cardiac slowing in response to blood pressure increase [4]. It was speculated that sympathetic control of heart rate might function as a vagus inhibitor rather than as an active cardiac accelerator. The authors admitted that cardiopulmonary baroreceptors could have been affected by changes in central blood volume, secondary to peripheral vasodilation or vasoconstriction, and that this might have altered arterial baroreceptor heart rate reflex.

The aim of our study was to investigate the effect of high thoracic epidural anaesthesia on baroreflex control of heart rate, in a controlled randomised trial, with an attempt to keep central blood volume equal in the different groups. Secondly, we compared the effect of two different bupivacaine concentrations (0.25% and 0.5%) on the baroreflex control of heart rate.

Methods

Thirty patients, with an ASA physical status of 1 or 2, scheduled for elective abdominal surgery entered the study. Subjects with any kind of cardiac or circulatory disorder, autonomic neuropathy, contraindications to epidural anaesthesia and contraindications to transoesophageal echocardiography were not studied. The abdominal operations included: duodenum-preserving resection of the

pancreatic head ($n = 12$), cholecystectomy ($n = 6$), cholecystojejunostomy ($n = 4$), liver tumour resection ($n = 2$), Whipple's pancreaticoduodenectomy ($n = 2$), resection of the sigmoid colon ($n = 1$), cholecystectomy + nephrectomy ($n = 1$), local resection of a papillary tumor ($n = 1$) and partial resection of the pancreas ($n = 1$). The patients were randomly assigned to group 1 (thoracic epidural with bupivacaine 0.25%, $n = 10$), group 2 (thoracic epidural with bupivacaine 0.5%, $n = 10$) or group 3 (control group without thoracic epidural, $n = 10$).

One of the patients assigned to the control group was not studied because echocardiographic recordings were of insufficient quality. Details of the patients are summarised in Table 1. The study was approved by the Human Ethics Committee of the University of Ulm and informed written consent was obtained from each of the individuals.

The epidural catheters were inserted on the evening before surgery in the patients in groups 1 and 2. The epidural space was punctured using the loss-of-resistance technique and the median approach. The site of insertion was the mid-to-lower thoracic region ($T_{7/8}$ – $T_{10/11}$).

All patients were premedicated with chlorazepate 20 mg orally 2 h before the haemodynamic measurements were taken on the day of the operation. Upon arrival in the operation theatre, intravenous cannulae, a radial artery catheter and ECG leads were placed. Doses of plain bupivacaine 0.25% and 0.5%, respectively, were injected into the epidural catheter until the upper level of the sensory block, evaluated by pin prick, exceeded T_1 .

In order to compensate for the peripheral vasodilation, a 6% hydroxyethyl starch solution was administered intravenously to patients in groups 1 and 2, but not to the control patients. The volume given was 10 ml.kg^{-1}

Table 1. Biometric data, extent of sensory block, volume and dosage of administered bupivacaine in groups 1, 2 and 3. All values are arithmetic means (SD); for the upper and lower levels of sensory block the range of segments are given in square brackets.

	Age (years)	Weight (kg)	Height (cm)	Epidural anaesthesia			
				Sensory block		Bupivacaine	
				Upper level (segment)	Lower level (segment)	Volume (ml)	Dose (mg)
Group 1, bupivacaine 0.25% (<i>n</i> = 10)	55 (12)	71 (21)	170 (11)	C8 [C ₇ -T ₁]	L2 [T ₁₂ -L ₄]	18.7 (3.5)	46.5 (9.0)
Group 2, bupivacaine 0.5% (<i>n</i> = 10)	51 (10)	67 (10)	172 (8)	C8 [C ₆ -T ₁]	L1 [T ₁₁ -L ₃]	16.8 (2.8)	84.0 (14.1)*
Group 3, control (<i>n</i> = 9)	43 (11)	66 (7)	168 (8)	—	—	—	—

**p* < 0.001 compared with group 1.

according to the results of a pilot study. To ensure equal volume loading in all three groups, the left ventricular end-diastolic area (LVEDA) was registered using transoesophageal echocardiography. This was performed after induction of general anaesthesia with midazolam 0.1 mg.kg⁻¹, fentanyl 5 µg.kg⁻¹ and vecuronium 0.1 mg.kg⁻¹. The trachea was intubated and ventilation of the lungs was controlled artificially with intermittent positive pressure ventilation and inspired N₂O of 60% in oxygen. The minute volume was adjusted to produce normocarbica (P_ECO₂ 4.6–6.0 kPa), (Capnometer Hewlett-Packard).

The transoesophageal echo probe (Diasonics 3400R Varian, 3.5 MHz transducer) was advanced to a position where left ventricular cross-sectional registrations at mid-papillary muscle level could be obtained [5]. Two-dimensional and M-mode registrations were displayed, together with arterial pressure curve, ECG and capnographic curve. All data were recorded simultaneously on videotape. Evaluation of the recorded haemodynamic data took place on an electronic device (Kontron, Cardio 200).

Haemodynamic measurements started when the extent of the sensory block included the segments T₁–T₅ and when stable haemodynamic conditions were present with the patient supine after volume expansion and induction of general anaesthesia. Baseline haemodynamic measurements included systolic and diastolic arterial pressure, heart rate and LVEDA.

The baroreflex sensitivity was assessed by measuring the heart rate response to an increase and decrease of systolic arterial pressure according to the method described by Smyth, Sleight and Pickering [6]. Intravenous doses of phenylephrine (1–2 µg.kg⁻¹) and nitroglycerine (0.5–1 µg.kg⁻¹) were administered in random order. The dose of each substance was selected to increase or decrease systolic pressure by 20–25%. The second pressure manoeuvre started when levels for systolic pressure and

heart rate returned to control values. R-R interval was plotted as a function of the preceding systolic pressure, starting with the onset of arterial pressure elevation and decline respectively. The slope of the linear portion of this sigmoid relationship (expressed in ms of R-R interval change per mmHg of systolic pressure change) was taken as baroreflex sensitivity. According to Bristow and co-workers we calculated a 'set-point' R-R interval at reference systolic pressure, which we defined as the mean baseline systolic pressure of the control group [7]. Only values obtained during expiration were included in our calculations.

The data on the linear portion of the systolic arterial pressure/R-R interval relationship were analysed using the least-squares linear regression analysis. Statistical evaluation of the demographic and haemodynamic data was performed using the one-way analysis of variance (ANOVA). The baroreflex slopes showed a skewed distribution and were subjected to ANOVA after logarithmic transformation of the data. For the parameters for which null hypothesis had to be rejected Scheffé's test was performed. A value of *p* < 0.05 indicated statistical significance. All values were expressed as arithmetic means (SD) with the exception of the values for baroreflex sensitivity, which were expressed as medians (95% CI).

Results

Although there were no significant differences between the groups in respect of their biometric data, the mean age in the control group was somewhat lower (NS) compared with groups 1 and 2. The volume of the administered local anaesthetic solution was equal in groups 1 and 2 as was the extent of the sensory block. As expected, the dose of bupivacaine was highest in group 2 (Table 1).

All haemodynamic data are summarised in Table 2. Systolic and diastolic arterial pressure as well as LVEDA measured prior to the beginning of the pressure

Table 2. Heart rate (HR), systolic (SAP) and diastolic (DAP) arterial pressure, left ventricular end-diastolic area (LVEDA), baroreflex sensitivity measure HR response to nitroglycerine (GTN-slope) and phenylephrine (PHE-slope) and R-R interval at reference SAP calculated from the nitroglycerine (N)- and phenylephrine (PHE)-regression equation, respectively. The values for GTN- and PHE-slope are medians (95% CI); all other values are given as arithmetic means (SD).

	HR (min ⁻¹)	SAP (mmHg)	DAP (mmHg)	LVEDA (cm ²)	Baroreflex			
					Sensitivity		Setpoint	
					GTN-slope (ms.mmHg ⁻¹)	PHE-slope (ms.mmHg ⁻¹)	GTN (ms)	PHE (ms)
Group 1, bupivacaine 0.25%	66 (12)	113 (15)	64 (10)	20.7 (7.3)	1.8 (1.3–2.9)*	6.8 (4.4–8.7)	971 (214)	993 (270)
Group 2, bupivacaine 0.5%	64 (10)	116 (13)	65 (10)	22.0 (6.5)	1.5 (0.7–2.9)*	4.5 (2.5–7.3)	961 (148)	919 (203)
Group 3, control	76 (12)	116 (12)	67 (6)	20.0 (5.8)	3.5 (2.7–6.5)	6.2 (3.6–9.3)	890 (253)	799 (176)

**p* < 0.01 compared with group 3 (control).

manoeuvres did not differ in the three groups. Heart rate in groups 1 and 2 was considerably lower than in group 3; however, the level of significance was not reached.

The baroreflex sensitivity measured as heart rate response to pressor manoeuvre was equal in all three groups. In contrast, the baroreflex slope in response to the nitroglycerine manoeuvre was significantly lower ($p < 0.01$) in groups 1 and 2, as compared with the control group. There were no differences between the two bupivacaine concentrations.

The R-R intervals at reference systolic arterial pressure calculated with the regression equations of the depressor manoeuvre, as well as those calculated with the regression equations of the pressor manoeuvre, showed no significant differences between the three, indicating that no resetting of the reflex occurred in either group.

Discussion

We found that sympathetic denervation of the heart by thoracic epidural anaesthesia markedly reduces cardio-acceleration in response to a decrease in arterial pressure, while preserving the slowing in response to increased arterial pressure. For none of the measured haemodynamic variables could a difference between the two bupivacaine concentrations be demonstrated. That suggests that an equal intensity of sympathetic block was achieved with either concentration. There was no resetting of the reflex with either bupivacaine concentration.

In our study, baroreflex function could potentially have been influenced by three mechanisms. General anaesthesia, whether it is performed with intravenous or inhalational agents, is known to alter baroreflex function [7–12]. We used transoesophageal echocardiography to assess left ventricular preload conditions because of the quality of its registrations. Introducing the oesophageal probe in awake subjects is unpleasant and could have caused autonomic reactions. Therefore, we chose to perform our measurements under general anaesthesia, which may have contributed to the generally low level of baroreflex sensitivity.

Epidural anaesthesia may influence the cardiovascular system either by block of afferent and efferent fibres or by the pharmacological effect of systemically absorbed local anaesthetic [13]. The latter effect may be twofold, firstly a direct depression of the cardiovascular system and secondly a stimulating effect, provoked centrally [14]. Although in our study there was no difference in the baroreflex function between the two bupivacaine concentrations, we cannot exclude some influence of absorbed bupivacaine.

The results of our present study are in contrast to those found by Takeshima and Dohi, who reported no change in baroreflex sensitivity evaluated with the depressor test and a suppression of the heart rate response to the pressor test [4]. Their investigation was performed on awake volunteers under cervico-thoracic epidural anaesthesia. It was suggested that in healthy subjects the sympathetic component of heart rate control modifies the dominating vagal tone rather than functioning as an active cardiac accelerator. This is in conformity with earlier investigations performed on healthy humans under beta-adrenergic, parasympathetic, and combined block [15, 16].

Results from the present study suggest that in our patients, tachycardic responses to decreasing arterial pressure were mainly transmitted by the sympathetic division of the autonomic nervous system. Cardiac slowing in response to increasing arterial pressure was not altered by cardiac sympathectomy and may therefore be attributed to parasympathetic activity. These findings are consistent with the traditional concept of Glick and Braunwald, who suggested that responses to increased arterial pressure pre-

dominantly involved vagal compensation, whereas responses to decreased arterial pressure were mediated by the sympathetic effector component [17]. One reason for the discrepancy between our results and those of Takeshima and Dohi could well be the fact that our data were obtained from anaesthetised patients. It is recognised that general anaesthesia may alter the balance between the baseline levels of sympathetic and parasympathetic activity [18].

It has been known for some time that afferent impulses from cardiopulmonary ('low pressure') receptors interfere with those from sino-aortic ('high pressure') baroreceptors in a way that the sensitivity of either reflex mechanism is augmented when the influence of the other mechanism is reduced or eliminated [19–23]. Unloading of cardiopulmonary receptors by epidural anaesthesia-induced decreases in systemic venous return have been shown to augment arterial baroreflex sensitivity for both pressor and depressor manoeuvre [24]. To minimise that influence in our study we expanded the intravascular volume in the thoracic epidural groups in order to equalise central blood volume in all three groups. Without that compensatory volume expansion we would possibly have obtained a smaller decrease in baroreflex sensitivity to the depressor test in the thoracic epidural groups. However, on the basis of our data we are not able to assess the potential influence of cardiopulmonary reflex mechanisms on the baroreflex control of the heart rate.

It can be concluded that in healthy humans under general anaesthesia, the baroreflex-mediated heart rate response to decreasing blood pressure is dependent on the integrity of the sympathetic nervous system. Whether the sympathetic nervous system functions as a direct cardio-accelerator, or indirectly by modifying the parasympathetic responsiveness should be the subject of future studies.

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Use of adrenaline in obstetric analgesia

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Summary

A questionnaire on the use of adrenaline in obstetric analgesia was completed by 87 obstetric anaesthetists: 71% of consultants in teaching hospitals were prepared to use adrenaline mixed with local anaesthetics compared with 33% of consultants in district hospitals; they had a similar duration of obstetric anaesthetic experience. Test doses containing adrenaline were not commonly used in labour, but were more often used prior to elective Caesarean section. Adrenaline was used with either lignocaine or bupivacaine; few consultants used both solutions. Contraindications to the use of adrenaline in the nonuser group were in decreasing order of rank: neurological damage, pregnancy-induced hypertension, stenotic valvular heart disease, sickle cell disease or trait and fetal distress. Overall, the contraindications related to the systemic absorption of adrenaline were most common.

Key words

*Anaesthesia; obstetric.
Anaesthetics, local.*

The addition of adrenaline to local anaesthetics for use in obstetric pain relief, whether as part of a test dose, or for the establishment of epidural analgesia is not universally accepted. Its addition to a test dose has been described to improve recognition of inadvertent intravascular injection [1], but in the context of labour, variability of heart rate changes may not produce clear signs of intravenous placement. Adrenaline can be added to local anaesthetics, especially for Caesarean section, to reduce systemic absorption, and hence peak blood levels and toxicity, to provide a longer duration of action and to intensify

blockade [2–4]. The clinical significance of effects related to uterine blood flow [5,6] and activity [7] is negligible, but it is during labour rather than at Caesarean section that these complications could be detrimental.

Where current practice is controversial, an analysis of the situation may be helpful. First, it will indicate which drugs and combinations are preferred so that consumerism may motivate their production. Second, the perceived indications and contraindications to the use of adrenaline can be defined for teaching and audit purposes. The opinion of a representative sample of members of the

Obstetric Anaesthetists' Association was chosen to obtain a range of views which would be prevalent throughout the United Kingdom.

Method

A questionnaire was distributed at the annual meeting of the Obstetric Anaesthetists' Association and the replies collected. Anaesthetists identified themselves either as consultants or anaesthetist-in-training, by their place of work (in a teaching or non-teaching hospital) and by the number of years practising obstetric anaesthesia. There were three areas of enquiry: the use of adrenaline in test doses, the preferred mixture of local anaesthetic and adrenaline, and particular contraindications to the use of adrenaline in obstetrics. The types and frequency of use of drugs mixed with adrenaline were sought and the indications for using a particular solution. A list of possible contraindications was given for selection and included fetal distress, pregnancy-induced hypertension, sickle cell disease or trait, stenotic valvular heart disease and neurological damage. The list could be extended by the respondent.

Where applicable, a Chi-squared test was used for statistical analysis.

Results

The questionnaire was completed by 76 consultants and 11 anaesthetists-in-training. The use of adrenaline by these groups is shown in Table 1. Seventeen consultants in teaching hospitals used adrenaline compared with seven who did not, whereas in other hospitals 17 consultants used adrenaline while 35 did not. This difference in use of adrenaline between teaching and non teaching hospital consultants is significant ($p < 0.01$). The majority of consultants had over 10 years experience in obstetric anaesthesia and the years of experience could not explain these differences of opinion.

Only two consultants used adrenaline solely in a test dose with bupivacaine, the remainder either administered it for the block, or both for the test dose and the block. The solutions used by these two latter groups differed. Anaesthetists who selected adrenaline for establishing epidural analgesia only, chose to mix a solution of lignocaine with the adrenaline (17 out of 21 anaesthetists), whereas those anaesthetists who administered adrenaline containing solutions with both the test dose and the block, chose the premixed solution of bupivacaine with adrenaline (13 out of 17 anaesthetists). This is a significant difference in practice ($p < 0.01$). The reasons for choosing adrenaline containing bupivacaine or lignocaine solutions were not clarified by some anaesthetists. Almost half the respondents in the bupivacaine and lignocaine user groups stated that

Table 1. The use of adrenaline in obstetrics among consultants and anaesthetists-in-training

	Consultants		
	District General Hospital	Teaching Hospital	Anaesthetists-in-Training
Nonusers	35	7	5
Users			
(a) test dose only	1	1	0
(b) test dose and epidural block	5	8	2
(c) epidural block only	11	8	4
Total	17	17	6

they used the adrenaline containing solutions for lower segment Caesarean sections to reduce toxicity and to achieve a better quality, more reliable block with a faster onset and more prolonged effect. One anaesthetist specifically stated that adrenaline may exert independent analgesia at the level of the spinal cord. This would relate to its effect at spinal alpha-adrenoceptors where it is antinociceptive[8]. Cautions to the use of adrenaline are listed in the *British National Formulary* [9]: these are ischaemic heart disease, diabetes mellitus, hyperthyroidism, interactions with anaesthetics, antidepressants and beta-blockers. There are no specific obstetric contraindications.

The response of anaesthetists to the contraindications presented in the questionnaire are recorded in Table 2. Three other contraindications were stated by individual anaesthetists; these were thyrotoxicosis, poorly established labour and the suggestion that the use of adrenaline containing solutions can increase the incidence of vomiting. The results have been divided into contraindications as recorded by user and nonuser anaesthetists. There are differences between these two groups. Many anaesthetists who avoid adrenaline specifically stated that this section of the questionnaire was not applicable to them, but they gave no reasons for their opinion which was often strongly expressed. Four, however, indicated that their main reason for not using adrenaline was its potential for neurological damage and left the other contraindications unanswered. One of these anaesthetists emphasised that neurological damage after the use of adrenaline containing solutions with the additional complication of hypotension following a sympathetic block may be indefensible. The potential for neurological damage was the main contraindication in the group of adrenaline nonusers. The other contraindications in order of perceived importance were pregnancy-induced

Table 2. Contraindications expressed by anaesthetists to the use of adrenaline in obstetrics. Two groups of anaesthetists are compared, those who use adrenaline with local anaesthetics and those who do not. 'Yes' indicates a positive response to a contraindication, 'No' a negative response and '?' either a not applicable comment or no answer.

	All anaesthetists n = 87			Adrenaline users n = 40			Adrenaline nonusers n = 47		
	Yes	No	?	Yes	No	?	Yes	No	?
Fetal distress	11 (13%)	42	34	7 (18%)	31	2	4 (9%)	11	32
Pregnancy-induced hypotension	27 (31%)	28	32	15 (38%)	24	1	12 (26%)	4	31
Sickle cell disease/trait	18 (21%)	34	35	11 (28%)	26	3	7 (15%)	8	32
Stenotic valvular heart disease	25 (29%)	27	35	14 (35%)	23	3	11 (23%)	4	32
Neurological damage	23 (26%)	33	31	10 (25%)	27	3	13 (28%)	6	28

hypertension, stenotic valvular heart disease, sickle cell disease or trait and fetal distress.

Fetal distress was considered by all anaesthetists to be the least important contraindication. Pregnancy-induced hypertension was overall the prime contraindication. Sickle cell disease or trait and stenotic valvular heart disease were significant contraindications in both groups. Comments were made by a few anaesthetists on the latter contraindication, that it would depend on the degree of the stenosis whether or not it was a contraindication. This response was recorded as positive. Some reservations were also expressed by a few adrenaline users on other contraindications.

Discussion

The group of obstetric anaesthetists canvassed in this study was mainly consultants of 10 or more years experience in the specialty. Teaching hospital consultants comprised 46% of the respondents which is probably higher than the national average. Their opinions on the place of adrenaline in obstetrics were biased towards its use, with 71% prepared to use it compared with 33% of district hospital consultants. It is not possible to explain this difference on the number of years since beginning training in the specialty. It could be postulated that changes in practice and training are occurring in the teaching hospitals, presumably in response to current research, which in future will be reflected in practice in the non teaching hospitals. Alternatively, techniques are being learned while in training which are not followed once a trainee has achieved consultant status. It is unfortunate that the number of anaesthetists-in-training in the sample was small and no conclusions can be drawn from their opinions.

Adrenaline was more often used with local anaesthetics to achieve epidural blockade rather than in a test dose. Where it was used in the test dose, some anaesthetists remarked that this was performed for Caesarean sections only. There was seen to be no indication for the use of adrenaline in the test dose during labour. This practice follows the clinical observations made in labouring women compared with the elective surgical cases [1, 10].

The choice of local anaesthetic to be used with adrenaline demonstrated two distinct groups of anaesthetists, one using bupivacaine and the other lignocaine but apparently for similar reasons. Both solutions are acidic, but bupivacaine is presented as a premixed solution with 1 in 200 000 adrenaline whereas the adrenaline and lignocaine mixture is prepared freshly by the anaesthetist because it has a short shelf life without preservative. Theoretically, a slower onset of action than the plain solution should be expected unless a pH modified solution is used. Lignocaine requires the addition of adrenaline to provide analgesia for Caesarean section, otherwise toxic effects may occur and adrenaline reduces lignocaine's absorption more than bupivacaine [3, 11]. If an anaesthetist has to mix a solution, it may be expected that the extra time needed, and the potential for inaccuracies and errors and subsequent liability to litigation would deter its use in clinical practice. This does not appear to be the case and so its use can be expected to continue.

The benefits of adding adrenaline to a local anaesthetic should be seen clearly if there is the chance of a serious complication occurring related to its use. Recent studies have shown improvements in the onset and efficacy of bupivacaine with adrenaline, both in labour and at Caesarean section, by the addition of opioids [12, 13] and alkalising solutions [14]. The use of adjuvants blurs the risk/benefit balance in a technique, but it also establishes the perceived need for an improvement in the technique. The adrenaline nonusers appear to consider that risks

outweigh benefits. The contraindications to the use of adrenaline are a measure of those risks.

The contraindications to adrenaline listed on the questionnaire may be a result of either adrenergic effects from accidental intravenous injection or from systemic absorption, or a local vasoconstrictor effect. An intravascular injection of 15 µg has been associated with fetal distress [15]. Few anaesthetists confirmed that fetal distress may be a contraindication, but perhaps this reflects its use mainly for Caesarean section rather than for labour. Rapid absorption from the epidural space has been demonstrated and in severe pre-eclamptic toxemia there is an increased sensitivity of patients to catecholamines [16, 17]. Pregnancy-induced hypertension was confirmed as the major contraindication to the use of adrenaline by all anaesthetists, and stenotic valvular heart disease was a close second.

Contraindications related to a local vasoconstrictor effect were sickle cell disease and neurological damage. One in three adrenaline users would not add adrenaline to the solution in patients with sickle cell disease. It may be a compounding factor to hypotensive spinal cord damage. Lumbar intrathecal adrenaline in dogs reduces lumbar dural blood flow but not spinal cord blood flow [18]. Spinal cord blood flow was measured after epidural lignocaine, keeping physiological and anaesthetic variables constant, and a small decrease in flow was measured with radioactive microspheres in the lower thoracic and lumbar region [19]. If this occurs in clinical practice together with sympathetically induced hypotension, neurological deficits should be more common unless the reduced blood flow reflects a decrease in spinal cord metabolic rate so that a degree of protection is provided to the spinal cord. The effects of local anaesthetics and their adjuvants on local regulation of spinal cord blood flow requires further study.

Serious neurological deficit is rare after epidural analgesia but spinal cord damage has been associated with high concentrations of adrenaline, hypotension [20] and irritant acid solutions [21]. Central nervous system damage can, however, follow acute toxicity to local anaesthetics with convulsions [22]. It is to prevent toxicity that adrenaline is administered with local anaesthetics [2, 3]. Hypotension from inferior vena caval occlusion and convulsions from eclampsia may further complicate the obstetric use of epidural analgesia. It was apparent from the replies obtained, but it could not be quantified, that adrenaline users were rarely applying the technique to all patients, so that a selection was often made as to which drug or drug combination should be used. This is probably where the balance between nonuse and careful use lies.

It will be interesting to review the changing pattern of use of adrenaline in obstetric analgesia in future years. Anaesthetists are not likely to change their opinions on the basis of a review of practice, but hopefully, this study will initiate a review of the reasons and contraindications for use or nonuse of adrenaline in obstetric units. Further information is required on training and subsequent techniques used.

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An evaluation of a combined spinal/epidural needle set utilising a 26-gauge, pencil point spinal needle for Caesarean section

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Summary

We have conducted a prospective study into the ease of use and incidence of postdural puncture headache with the 'Portex' combined spinal/epidural set. The pack contains a 16-gauge Tuohy needle of standard 8 cm shaft length with a matching 26-gauge pencil point spinal needle. The study included 150 consecutive combined spinal/epidural anaesthetics for lower segment Caesarean section. Eighty-eight percent of the cases fulfilled the criteria as technically perfect, i.e. cerebrospinal fluid obtained at the first attempt after identifying the epidural space. There were two cases of significant postdural puncture headache requiring blood patch due to puncture by the 26-gauge spinal needle giving an incidence of 1.3%. This compares favourably with previously reported rates in obstetric patients.

Key words

Anaesthesia, obstetric.

Anaesthetic techniques regional; epidural, spinal.

The place of spinal anaesthesia and combined spinal/epidural (CSE) anaesthesia in obstetric practice has been reviewed in recent months [1, 2]. The greater speed of onset and better quality of analgesia achieved with subarachnoid

block for Caesarean section has found favour with many anaesthetists and is now the commonest technique used in the USA [3]. The ability to refine the analgesia with epidural supplements and extend the analgesia well into the

postoperative period is making techniques combining spinal and epidural anaesthesia increasingly popular.

One of the principal concerns with regard to subarachnoid blockade in obstetric patients is an incidence of postdural puncture headache (PDPH) as high as 14.5% or 9.5% [4, 5] even with needles as small as 25 gauge or 26 gauge. PDPH is particularly debilitating in the nursing mother because of its postural nature [6].

Attempts to reduce the incidence of PDPH have centred around the use of very fine needles, and more recently the return to favour of needles with pencil point or atraumatic tips [7]. Although there are reports of high success rates with cutting-tipped needles as small as 29 gauge using a CSE technique, where the epidural needle acts as a guide to the spinal needle [8], such fine needles are generally considered difficult to use [9]. The popularity of needles with atraumatic or pencil point tips [10–13] is due to the possibility of using larger gauge needles of this design without increasing the incidence of PDPH. The 'Portex' Single Use Spinal/Epidural Set comprises a 16 gauge Tuohy needle (external diameter 1.65 mm) with an 8 cm shaft and a 26 gauge pencil point spinal needle (external diameter 0.46 mm, internal diameter 0.23 mm) with a 11.7 cm shaft and transparent plastic hub (Portex, Hythe, UK, Ref. 100/396/816). This gives 1.2 cm of spinal needle protrusion past the Tuohy needle tip when fully inserted. A pilot study of 38 patients using this needle set resulted in no cases of PDPH [14]. We have further assessed this equipment for its technical ease of use by both senior and trainee staff and the incidence of PDPH.

Methods

One hundred and fifty consecutive women due for lower segment Caesarean section (LSCS) in which the mother had chosen to be awake were studied. The procedure was explained and the usual contra-indications to regional anaesthesia applied.

Patients were positioned in a modified left lateral position [15]. A minimal head-down tilt ensures that sufficient hyperbaric spinal solution reaches the mid and upper thoracic nerve segments. In addition, an inflated 3 l 'Polysusor' bag under the shoulder and three pillows under the head provides lateral flexion of the upper thoracic and cervical spines. This reproduces the uphill gradient to the upper thoracic and cervical segments normally present in the supine position, thus protecting these high nerve roots from blockade by the hyperbaric spinal solution. This position has the additional benefits of minimal aorto-caval compression, and of 'trapping' the hyperbaric solution in an appropriate (thoracic) portion of the subarachnoid space should there be any delay introducing the epidural catheter. After the latter had been inserted the patients were turned into the identical right lateral position. The supine position with inflatable wedge under the right hip was not adopted until surgery was about to start.

Additional prophylaxis against hypotension comprised the use of elastic support stockings, preloading of the circulations of all patients with compound sodium lactate solution (1500 ml) and an infusion of ephedrine (50 mg in 500 ml of compound sodium lactate solution) titrated against the blood pressure [16]. Hyperbaric 0.5% bupivacaine 2.8 ml was used in all patients, irrespective of height. Although not detailed in this study, significant hypotension with this technique was unusual.

Technical problems encountered during the technique were recorded and the procedure was graded as 'technically perfect' or 'imperfect'. A perfect procedure required that after identifying the epidural space with the Tuohy needle, that the dura was punctured at the first attempt with the

spinal needle, that there was free backflow of cerebrospinal fluid (CSF) and that this was easily aspirated at the start and end of the local anaesthetic injection.

It was also noted whether or not a sensation was felt as the spinal needle emerged from the Tuohy needle or as it pierced the dura. The time to appearance of CSF was recorded as was the level of spinal block and the need for any pre-operative epidural supplementation. This was given to any patient in whom bilateral analgesia to skin pinching did not reach as high as T₄.

Postoperatively the mother received two epidural top-ups for analgesia when requested, each usually comprising 10 ml of solution (6 ml normal saline, 2 ml 0.5% bupivacaine and 100 µg fentanyl). During this time she was kept in a recovery ward. The epidural catheter was then removed and the mother sent to the postnatal ward. She was visited during the days prior to discharge and questioned about headache, neckache or other neurological symptoms.

Results

Of the 150 cases studied 106 were for elective and 44 were for emergency LSCS. Sixty five of the procedures were performed by a consultant anaesthetist and 85 by trainee anaesthetists (Table 1).

Anaesthetics were classified as technically perfect in 88% of all cases (89% for elective surgery and 86% for emergency cases). The paramedian approach was used in 121 of the anaesthetics but there was no statistical difference between that approach and the midline approach for the incidence of a perfect technique.

The mean time to the appearance of CSF in the spinal needle hub was 6 s and the sensation of piercing the dura was felt in 93% of cases.

A technically perfect procedure was performed in 92% of cases by a consultant anaesthetist as compared to 86% amongst trainees. This difference did not reach statistical significance.

For the purposes of this study a 'significant' PDPH was defined as a postural headache occurring after dural puncture that was (a) continuous for more than 24 h at any level of intensity or (b) so severe at any time that the patient was unable to maintain an upright posture, i.e. any postural headache that had a significant effect on the mother's postoperative well-being.

Three mothers developed a significant PDPH for which treatment by epidural blood patch was required. One of these occurred following a dural tap with the Tuohy needle. Six other patients were noted to have a postoperative headache, but these were mild, nonpostural and self-limiting.

One anaesthetic was a complete failure and the mother received a general anaesthetic. Of the remainder, 28%

Table 1. Number of cases satisfying 'perfect' and 'imperfect' criteria.

	Technique	
	Perfect	Imperfect
Consultant anaesthetist	60 (92%)	5
Trainee anaesthetist	73 (86%)	12
Paramedian approach	107 (88%)	14
Midline approach	26 (90%)	3
Elective LSCS	95 (89%)	11
Emergency LSCS	38 (86%)	6

LSCS, lower segment Caesarean section.

received epidural supplementation prior to surgery to extend the block up to at least the T₄ level. No woman required any perioperative intravenous, inhalational or epidural supplementation.

Discussion

These results demonstrate that the CSE technique provides reliable regional anaesthesia for LSCS (0.67% failure rate). The single complete failure to achieve analgesia was interesting as it had in all other respects been a 'perfect' technique. The subarachnoid space was identified by free flow of CSF and confirmed by aspiration at the start and end of injection. Despite this, no anaesthetic dermatomal level was identified. As the anaesthetic ampoule was confirmed to be 0.5% heavy bupivacaine, it can be concluded that despite the appearance of CSF, the local anaesthetic was not injected into the subarachnoid space. Others have reported a similar occurrence using Sprotte needles [17] and it has been suggested that with pencil-point needles, if the diameter of the lateral orifice is large enough it may straddle the dura. As the thickness of the dura in the lumbar region may be as little as 0.5 mm [18] this may happen with any needle where the diameter of the needle hole in its long axis significantly exceeds this measurement. As the diameter of the 'Portex' needle orifice is slightly more than 1 mm, and of the 22 gauge or 24 gauge Sprotte needles nearer 2–3 mm, with these needles CSF may be aspirated from the distal part of the hole whilst the injection might flow predominantly via the proximal part into the epidural space with minimal analgesic effect.

The high rate of perfect techniques by both consultants and trainees, some of whom were new to the technique, suggests that this is a reliable method for routine practice and teaching. Some anaesthetists were sufficiently confident to use it in emergency LSCS, again with good results.

The comparative lengths of the two needles is such that the hub of the spinal needle usually rests against the hub of the Tuohy needle, thus facilitating a steady hold for injection.

The results show that three of the patients in this study had a headache severe enough to warrant epidural blood patching. One of these occurred after a dural tap by the Tuohy needle in a woman with a remarkably superficial epidural space. If this case is excluded the incidence of PDPH associated with the 26 gauge pencil point needle is 1.3%, while that resulting from the CSE technique as a whole is 2%.

Of the two other women requiring a blood patch, one was seen and received a blood patch without the knowledge of the investigators. When questioning the patient retrospectively, it appeared that although she had a headache which was relieved by a blood patch, it did not show the characteristic feature of PDPH in being relieved by the head-down posture and she was pyrexial. However, this case was included as a PDPH.

The low incidence of PDPH in this series compares favourably with previously published results using cutting-tipped needles [4, 5]. There are several possible explanations for this, but the needle design must be the most important. In addition, it has been shown that CSF leakage through punctured dura may be reduced by an oblique passage of the needle through the fibres [19]. The needles used in this study are such that the spinal needle emerges at an angle of approximately 4–5° from the long axis of the Tuohy needle. The paramedian technique, which tends to approach the dura at an oblique angle, was used in 81% of cases. If the orifice of the Tuohy needle is facing cephalad then these two factors combine to give a more oblique puncture of the dura.

Secondly, all the patients in the study received two 10 ml epidural injections of combined local anaesthetic and opioid. The epidural injection of saline boluses [20] and of opioids [21] has been recommended as prophylaxis against PDPH after accidental dural puncture, although the boluses recommended were of 60 ml.

In summary, we found the use of this equipment readily learnt by both senior and trainee anaesthetists, with a high percentage of technically perfect blocks and a low incidence of postdural puncture headache.

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Modified parallel 'Lack' breathing system for use in dental anaesthesia

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Summary

A twin-tube breathing system for inhalational anaesthesia in dental surgery is described. The system is a modification of a parallel Mapleson 'A' breathing system and is suitable for use with continuous flow anaesthetic machines. Resistance to airflow has been evaluated and is within the recommended ranges at all flows likely to be experienced in normal clinical conditions. The system is suitable for children and adults, easy to use and efficient. The expiratory valve is located remote from the face and the system is suitable for scavenging by active, assisted or passive systems.

Key words

*Equipment; breathing systems, parallel Mapleson 'A'.
Anaesthesia; dental.*

The important features of a breathing system suitable for dental chair anaesthetics are that it should be compatible to the equipment available, not require major changes in techniques of anaesthesia, be easy to clean and maintain and have a safe gas disposal system [1]. Furthermore, it should be suitable for a wide range of age groups.

It has long been held that breathing systems incorporating an expiratory valve, especially one remote from the facemask, are unsuitable for paediatric use [2]. More recently, the Humphrey ADE breathing system, comprising lightweight low resistance tubing and a low resistance expiratory valve, has been recommended as suitable for children [3,4]. In the 'A' mode, this is essentially a parallel Mapleson 'A' system.

We describe a breathing system for use with continuous flow machines, which is suitable for dental chair anaesthesia. We have evaluated the inspiratory and expiratory resistance of the system, efficiency, use in all age groups and suitability for scavenging under laboratory and clinical conditions.

Method

Apparatus. The system, a modification of a parallel Mapleson 'A' breathing system (MIE, Exeter) (Fig. 1), comprises: an apparatus-end, consisting of the parallel breathing system adaptor and two 150 cm lengths of 22 mm corrugated disposable tubing; a patient-end, consisting of two 15 cm PVC tubes (internal diameter 9 mm) adapted to connect to the corrugated tube and nasal inhaler (McKesson rubber nasal inhaler with expiratory port blanked).

Fresh gas is delivered via the afferent limb which contains a 2 l reservoir bag. Expired gas is returned to the adaptor via the efferent limb and discharged through an adjustable exhaust valve with a 30 mm tapered outlet (MIE, 'Superlight').

Experimental investigation. The pressure-flow characteristics of the system were investigated using the apparatus shown in Figure 2. Air was supplied from a piped source and the flow controlled by a needle valve and measured by a flow meter. Each flow was checked with a calibration analyser (Timeter Instrument Corporation RT200). The air

was passed via a 22 mm male connector (incorporating a side port for pressure measurement) into the breathing system. The side port was connected by a tube to one limb of a U-tube water manometer, the other limb of which was open to the atmosphere.

The breathing system was tested in both inspiratory and expiratory modes. In addition, the apparatus was tested in the expiratory mode with a 5 m length of 22 mm corrugated exhaust tube attached to the exhaust valve to evaluate the effect of passive scavenging.

All the tests were conducted at 22°C with the apparatus laid out straight and unstretched on a bench. Flows of 10–90 l.min⁻¹ in ascending then descending steps were used. Each reading was taken after allowing the pressure in the apparatus to stabilise for at least 30 s. The reading at each step was repeated three times and the average of the sets of readings taken.

Clinical investigation. The apparatus was tested for rebreathing on 23 consecutive patients of ASA 1 or 2 status presenting for outpatient dental surgery (Table 1). End-tidal carbon dioxide ($PE'CO_2$), inspired carbon dioxide ($PICO_2$) and respiratory rate were monitored using a Datex Capnomac II capnometer, sampling gas from a site 5 cm

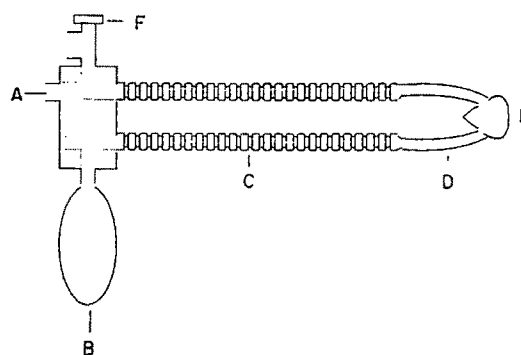


Fig. 1. Diagram of the 'MIE' parallel breathing system modified for dental anaesthesia. (A) gas inlet; (B) reservoir bag; (C) 22 mm corrugated tubing; (D) 9 mm PVC tubing; (E) nasal inhaler; (F) adjustable exhaust valve with 30 mm adaptor.

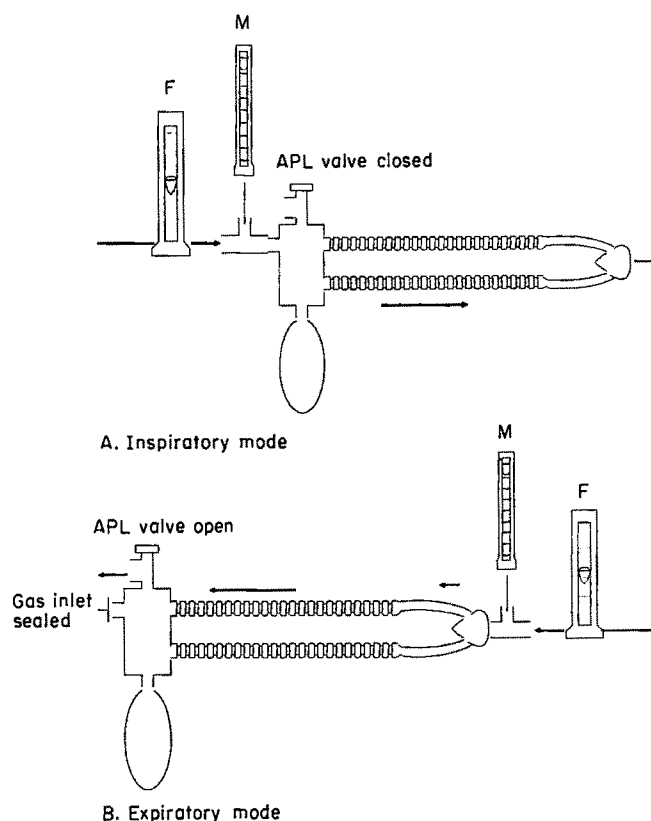


Fig. 2. Apparatus set up for measuring pressure changes produced by airflow through the breathing system. (F) flowmeter; (M) water manometer. Arrows indicate direction of airflow.

into the efferent limb of the system. This has been shown to be the optimum sampling site in afferent reservoir systems to detect rebreathing [5]. The capnometer was calibrated against a known standard before the study.

Induction of anaesthesia was either with methohexitone or by inhalational oxygen, nitrous oxide and halothane. Anaesthesia was maintained with nitrous oxide and halothane in 30% oxygen. The fresh gas flow was initially high until the reservoir bag was distended and the anaesthetist satisfied with airway control; it was then reduced to 3 l.min^{-1} for children under 20 kg and $150 \text{ ml.kg}^{-1}.\text{min}^{-1}$ for larger patients. Surgery was commenced when the anaesthetist considered that the depth of anaesthesia was sufficient. The duration of anaesthesia was taken as the time from induction to completion of surgery.

Respiratory rate, P_{ICO_2} and $P_{\text{E}'\text{CO}_2}$ were recorded immediately prior to commencement of surgery and for the remainder of the procedure. Pulse oximetry was used throughout.

Results

Resistance. The pressure changes in the system produced by air flow in the inspiratory and expiratory directions, with and without scavenging tubing attached, are shown in

Table 1. Patient characteristics in the study group; mean (range).

<i>n</i>	23
Age; years	9.5 (3–45)
Weight; kg	29.4 (13–72)
Duration; min	4.5 (3–9)
M:F	10:13

Figure 3. The results were obtained with the adjustable pressure limiting (APL) valve closed in the inspiratory mode and fully open in the expiratory mode.

Fresh gas flow and rebreathing. The respiratory rate, P_{ICO_2} and $P_{\text{E}'\text{CO}_2}$ are summarised in Table 2. Baseline values were recorded immediately prior to commencement of surgery and the peak respiratory rate and P_{ICO_2} at the end of the procedure were noted. In 11 patients, the P_{ICO_2} increased above zero and in all cases the $P_{\text{I}'\text{CO}_2}$ either remained at the baseline level or decreased. The oxygen saturation was above 95% throughout.

Discussion

Martin, Kong and Lewis studied resistance to airflow in a standard parallel Mapleson 'A' (Lack 2) system [6].

Table 2. Respiratory rate, end-tidal and inspired partial pressures of carbon dioxide during the procedure; mean (SD).

	Respiratory rate	$P_{\text{E}'\text{CO}_2}$	P_{ICO_2}
Values at start of surgery	13.5 (3.5)	5.92 (0.40)	0
Values at the end of the procedure	22.5 (6.5)	5.43 (0.26)	0.1 (0.13)

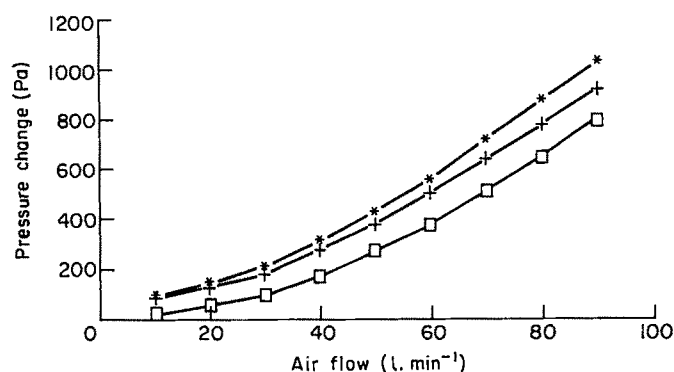


Fig. 3. Pressure differences produced by airflow through the breathing system. □, inspiratory; +, expiratory; *, expiratory with scavenging tube.

Expiratory resistance was found to be similar to our system, but inspiratory resistance was significantly lower. This confirms that breathing systems should be studied as a whole when considering their resistance to breathing. The total resistance of the system is the result of a complex interaction between adjacent components.

Nunn has suggested that pressures greater than 3 cmH₂O (294 Pa) at flows at 30 l.min⁻¹ and 6 cmH₂O (588 Pa) at flows of 60 l.min⁻¹ represent excessively high resistances [7]. The system described falls within this range. It must be remembered that in the anaesthetised dental patient, resistance is more likely to be due to obstruction at the nasal inhaler or due to poor insertion of the mouth-pack. Technique is of major importance.

Previous studies to assess the fresh gas flow at which rebreathing occurs have concentrated on achieving stable conditions before measurement began [8]. We feel that the short duration of most dental chair procedures makes this impractical.

Rebreathing in children with the Humphrey ADE system in 'A' mode has been shown to occur at a mean fresh gas flow of around 125 ml.kg⁻¹.min⁻¹, and Waaben *et al.*, investigating a co-axial Lack system for use in dental anaesthesia, found that no rebreathing occurred at flows of 150 ml.kg⁻¹.min⁻¹ [1,3,4]. We chose to evaluate the incidence of rebreathing under clinical conditions at 150 ml.kg⁻¹.min⁻¹ with a minimum flow of 3 l.min⁻¹.

Before surgical stimulation, all patients had an P_{iCO_2} of zero, indicating no rebreathing. On commencement of surgery, most patients exhibited a rise in respiratory rate. Eleven patients exhibited a rise in P_{iCO_2} (max 0.4 kPa) signifying rebreathing; in all these patients, respiratory rate increased and $P_{E'CO_2}$ remained decreased (Fig. 4).

The Mapleson 'A' system is very efficient in spontaneous respiration, requiring fresh gas flows close to predicted alveolar ventilation to prevent rebreathing [9]. Alveolar ventilation correlates poorly with body weight in children

and also varies with the depth of anaesthesia and degree of surgical stimulation. We feel that the rebreathing exhibited by these patients was a result of the alteration in the ratio of alveolar ventilation to fresh gas flow occurring in lightly anaesthetised patients. This being the case, and in the absence of a significant increase in metabolic rate, the P_{aCO_2} (and $P_{E'CO_2}$) will not increase and so is of little practical consequence.

Pollution during dental chair anaesthesia is a significant problem and derives from three main sources: nosepiece leakage, mouth-breathing and the expiratory valve [2]. The first two may be solved by meticulous technique, the latter by effective scavenging. Scavenging in most systems used for dental chair anaesthesia is from an expiratory valve positioned on the nasal inhaler, tending to increase the bulk of the apparatus. In the system described, the expiratory valve is remote from the nosepiece and lends itself to active, semi-active or passive scavenging. Five metres of 22 mm corrugated tubing results in minimal increase in expiratory resistance and may be lessened by the use of 30 mm tubing. The relative efficiency of the system further aids pollution control.

The system described is lightweight and easy to use. The nasal inhaler with valve removed is relatively unobtrusive. It is efficient, easy to scavenge from and suitable for use in all age groups presenting for dental chair anaesthesia.

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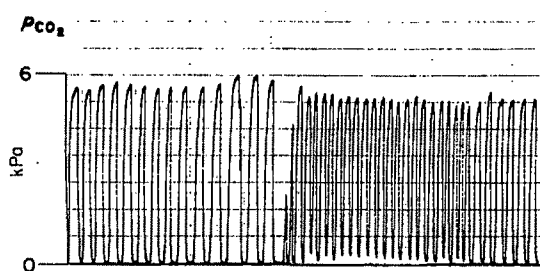


Fig. 4. Capnograph trace showing the changes in respiratory rate, P_{iCO_2} and $P_{E'CO_2}$ occurring with surgical stimulation.

The Gulf war: anaesthetic experience at 32 Field Hospital Department of Anaesthesia and Resuscitation

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Summary

The organisation and the workload of the anaesthetic department of a Field Hospital deployed during the Gulf war were described. Suggestions are made as to how the problems encountered might be overcome in the future.

Key words

Organisations; armed forces

Medical support in war is deployed as four echelons [1]. The casualties normally receive initial life-saving first-aid treatment at the first line Regimental Aid Post. The Field Ambulance, as second line, provides facilities for collection and treatment of casualties before evacuation to the third line, Field Hospital, where resuscitation and emergency life saving surgery are undertaken. The injured are then evacuated to the General Hospital (Fig. 1). The 32 Field Hospital, RAMC (32 Fd Hosp) was a 200 bedded unit staffed by the regular strength of the British Military Hospital Hannover, reinforced by regular personnel from all three Services, the Territorial Army and Reservists.

On the 17 January 1991, when the air war began, 32 Field Hospital was a collection of stores, freight containers and tents. Over the next few days, personnel and additional stores were brought 400 km up the main supply route from Al-Jubail to the operational location in the desert west of the Wadi Al-Batin (Fig. 2). Intense unpacking of stores and rapid erection of tents followed and the hospital was operational on 21 January. Casualties were triaged on arrival and channelled either to the treatment department or the resuscitation department depending on the nature and severity of their injuries [2]. This article describes the experiences of the Resuscitation and Anaesthetic departments of 32 Fd Hosp.

Resuscitation department

The resuscitation department of a Field Hospital must be able to deal with large numbers of seriously injured casualties arriving simultaneously or in quick succession. Efficient casualty handling is influenced by the layout of the department, availability of supplies and by the performance of the resuscitation personnel. They must be able rapidly to identify and treat those conditions that pose the greatest threat to life. All staff were therefore trained in the concept of British Army Trauma Life Support (BATLS) which is the military modification to the Advanced Trauma Life

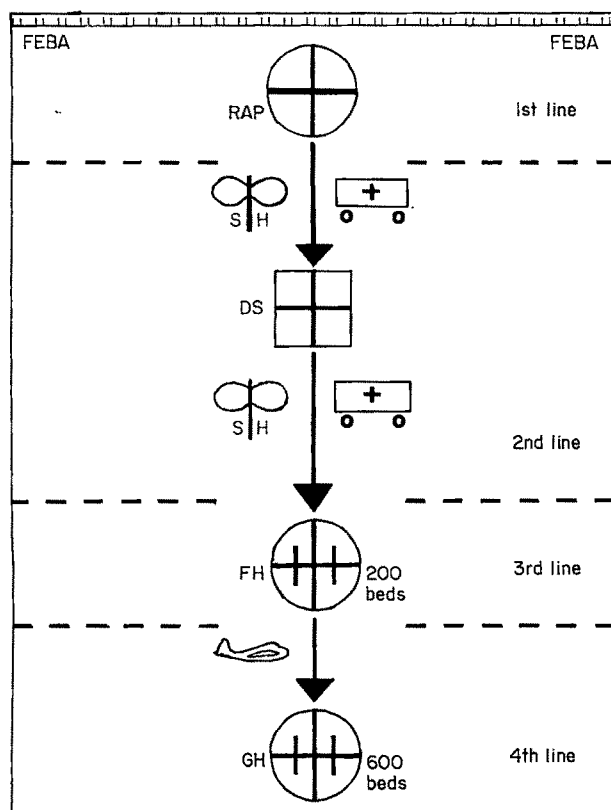
Support system [3]. This system of horizontal management minimises the times required for stabilisation before surgery, and identifies those patients who require immediate surgery as a part of the resuscitation process [4]. The team, consisting of a medical officer, a nursing officer and a combat medical technician, were given the same training and were able to exchange roles as the need arose. This allowed the medical officer a degree of freedom to maintain an overview of each case ensuring the effectiveness of individual treatment and interventions. The system may surprise civilian trauma practitioners, since it involved considerable delegation of clinical responsibility to the non-medical members of the team. Strict adherence to the BATLS drill ensured that no injuries were missed, and allowed for accurate and reproducible documentation.

Operating theatres

The operating theatre complex was an open tented area containing eight field operating tables (McVicar) and a central sterilising facility. Two further operating tables were sited in a collective chemical protection area (COLPRO). All tables were equipped with the Triservice anaesthetic apparatus [5]. A ventilator (CAPE Tc50) replaced the self-inflating bag for ventilation. A syringe infusion pump (Ohmeda 9000) and a manual sphygmomanometer were available at each table. Two automatic noninvasive blood pressure monitors (Critikon Dinamap) and four pulse oximeters (Critikon, Dinamap) were shared between the eight tables. The monitors were allocated either to those tables designated to receive the more severe casualties or to those staffed by relatively junior anaesthetists. Anaesthetic staffing consisted of three Consultants, three Senior Registrars, one Registrar and two Senior House Officers, although two Consultants and the SHOs worked in the resuscitation department. Hence, three of the operating tables were each manned by a combat anaesthetic support officer (CASO). The General Duty dental

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SH, support helicopters; RAP, regimental aid post; DS, dressing station; FH, field hospital; GH, general hospital.

Fig. 1. The chain of evacuation of a casualty from the forward edge of the battle area (FEBA).

surgeons of the Royal Army Dental Corps undergo basic anaesthetic training during peace time for their war role as CASOs. They require close and constant supervision. Four anaesthetic stations were created by arranging the operating tables in pairs of V formation. This allowed close supervision of the CASOs. Because of the threat of chemical attack a small part of the hospital was set up in a toxic-free environment. The COLPRO consists of units of tents lined inside with chemical proof fabric. Air filtration units pump toxic agent-free air to inflate the liners. In effect, they form units of bubbles. These can be joined together by connecting air locks and corridors. Air locks keep out toxic agents providing a clean environment.

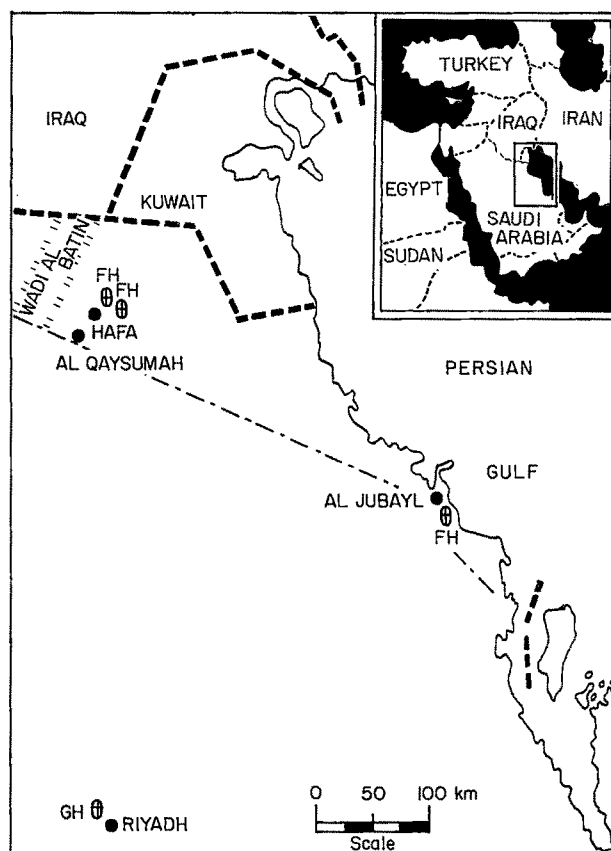
Regular hospital exercises were run to test the hospital and departmental procedures. Before the ground war, anaesthetics were administered for injuries sustained during training activities. This proved to be invaluable for identifying problems.

One hundred operations were performed in the period 21 January–6 March. The anaesthetic techniques used were: 91 general anaesthesia; seven local infiltration and regional blocks, one spinal and one sedation.

The frequency distribution of operating and recovery times are shown in Figures 3 and 4, and refer to the length of time the casualties remained in each department.

Discussion

The anticipated high battle casualty load did not occur. The hospital treated just under 1200 patients of all types.



FH, field hospital; GH, general hospital.

Fig. 2. Map of the area.

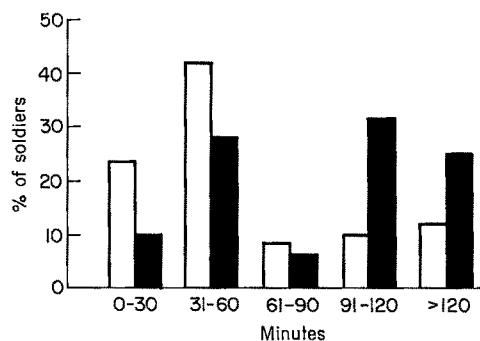


Fig. 3. Frequency distribution of operating times. □, Allied soldiers; ■, Iraqi soldiers.

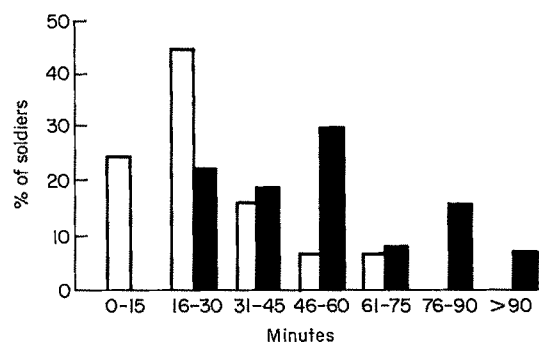


Fig. 4. Frequency distribution of recovery times. □, Allied soldiers; ■, Iraqi soldiers.

Only one postoperative death occurred due to haemorrhage in an intracranial injury.

General anaesthesia using inhalational techniques was the most common method used. Total intravenous anaesthetic techniques were not used frequently because of the relative inexperience of most of the anaesthetic department with this system [6]. Thiopentone remained the safest and most flexible of the induction agents. Ketamine was used in patients with persisting hypotension. Halothane was the only agent freely available in the field. Isoflurane was available in limited quantities and was reserved for those patients with suspected head injury. Although trichloroethylene is not commercially available for medical use in the United Kingdom, our supply was from the war mobilisation stock.

The large number of limb injuries presented some problems with siting of intravenous cannulae, blood pressure monitoring and pulse oximetry, all of which often had to be carried out on one limb. Leg cuffs for monitoring blood pressure and ear probes for oximetry would have helped. The multiplicity and the generalised distribution of injuries required frequent repositioning of the patients on the operating table. Optimal positioning of the casualty was often difficult on the stretcher-topped field operating table.

The length of surgery and of the stay in recovery area deserve comment. The long duration of surgery was due to the multiple injuries associated with the large number of preformed metal fragments that are a characteristic of modern ordnance. The time that casualties stayed in the recovery area was longer than usual and reflected prolonged recovery. This was multifactorial in origin. The use of halothane, trichloroethylene, morphine and papaveretum may have contributed. Perhaps agents with faster clearance would be more appropriate. The clinical use of isoflurane and enflurane has been compared with halothane in the Triservice anaesthetic apparatus. Significantly shorter recovery times have been recorded with enflurane [7] but not with isoflurane [8]. The Iraqi prisoners of war were all undernourished, dehydrated, and had been starving for several days before admission. The time between moment of injury to treatment was also longer than that of the allied casualties. Saraiva *et al.* [9] found that recovery time was significantly prolonged in mice which had low net protein utilisation and decreased total and dry body mass following intravenous anaesthetic agents. Ethnic differences in clinical response to opioids may also be a factor [10, 11]. Because of the low number of casualties, the operating theatre and recovery department were never overwhelmed. However, where there are mass casualties with limited recovery facilities a rapid return to consciousness after anaesthesia is imperative. At the time of deployment the Field Hospital had no facilities for intensive care or postoperative ventilatory support. Consequently, operating tables were occasionally blocked while waiting for return of spontaneous respiration.

The threat of chemical attack posed its own problems. In the conventional operating theatre and in the event of an imminent chemical attack the anaesthetised casualty would be protected from the inhalation of chemical vapour by the attachment of a chemical agent absorption filter at the air intake port of the ventilator. Pretreatment for nerve agent poisoning with pyridostigmine causes a 20–40% reduction in acetylcholinesterase activity [12] which interferes with the duration of action of muscle relaxants. However, no such interference has been reported with alcuronium [13]. The COLPRO (when closed down for protection against chemical agents) has many problems. These include isolation from natural environment, claustrophobia and poor temperature control, all of which may cause physical and

mental degradation of function. Communication between various departments is also difficult. Logistic problems are lack of space, poor lighting and transport of casualties. An unusually high concentration of anaesthetic vapour pollution also occurs [14].

The outside environment posed specific problems, particularly the air temperature, which varied from 40°C to –2°C. Sand storms and flooding caused difficulties with maintaining cleanliness in the operating theatre.

Finally, recommendations are proposed as to how the problems encountered might be overcome in the future. A major factor in the success of the BATLS system practiced at 32 Field Hospital was that 50% of the medical officers employed in the resuscitation department were anaesthetists. Anaesthetists, by the very nature of their training are the most effective practitioners of trauma resuscitation. At present, there is no official policy to deploy anaesthetists to the Resuscitation Department and this is left to the discretion of the Unit Commanding Officer. It is recommended that all medical officers in the resuscitation department be anaesthetists. While the open-plan tented operating theatre was spacious and provided a good working atmosphere, extremes of temperature, flooding and sandstorms caused problems. The deployment of a portable solid wall operating theatre might at least avoid some of these. The stretcher-topped field operating table, with its advantages of easy and rapid handling of casualties, does not allow optimal positioning or head-down tilt in an emergency. Hard-topped tables with these facilities are preferable. The field hospital should include a facility for an intensive care/high dependency unit. The existing ground role resuscitator (PNEU PAC 500-A764) consisting of up to 10 ventilators could be used for ventilatory support. The lack of patient monitoring equipment concerns us. A pulse oximeter, ECG, noninvasive blood pressure apparatus and a disconnection alarm are considered to be minimum requirements. Adequate provision must also be made for recovering patients.

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J.M. Handel, MRCP, FRCAnaes and N.J.H. Davies, DM, MRCP, FRCAnaes		Nil by mouth after midnight	1011
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Basic life support training

We share the disappointment of Dr Elias (*Anaesthesia* 1992; 47: 357), who demonstrated that 14 junior anaesthetists failed to meet the criteria set by the American Heart Association for successful cardiopulmonary resuscitation (CPR) and basic life support (BLS). Severe inadequacies in the CPR skills of nurses and house officers have also been reported [1, 2]. It is known that resuscitation experience without feedback increases confidence, not skill [3].

Recently, Morris *et al.* demonstrated improvements in theoretical knowledge with little improvement in skill of BLS in a 5-year follow-up of an original paper [4]. Why are current levels of teaching, assessment and retraining inadequate? Is therefore the basic care of the sick or obtunded patient inadequate?

To address these questions we conducted a postal survey of 30 University Anaesthetic Departments to define current

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Table 1. Teaching of BLS skills in University Hospitals.

	Students	House staff
Cardiopulmonary resuscitation course	95%	80%
Duration of cardiopulmonary resuscitation course	2-6 h	1-2 h
Refresher course	50%	20%
Assessment—written	25%	0
—viva	20%	0
—practical	60%	40%
—none	40%	60%
Staff/pupil ratio (range)	5.8 (2-10)	8.4 (2-20)
Number of manequins (range)	3.7 (1-12)	2.5 (0-6)
Tracheal intubation taught	80%	60%
Compulsory course	60%	35%

practice in the teaching of resuscitation and anaesthesia skills. The 22 completed questionnaires, a response of 73%, were evaluated and the results are summarised in Table 1. All hospitals run resuscitation courses for undergraduate medical studies, but only 80% for housemen. Refresher courses were run by only 33% and 20% for students and house staff respectively. No formal assessment of BLS skills was conducted by 40% of hospitals for students and 60% of hospitals for house staff. Tracheal intubation was taught on a course by 80% for students and only 60% for house staff. Only 25% of hospitals provided a resuscitation course for senior doctors and only 50% provided a resuscitation officer.

Airway management is basic in the care of the anaesthetised patient and important in the teaching of BLS. The anaesthesia component of the undergraduate curriculum varied from 1 to 4 weeks. The ideal duration of undergraduate anaesthesia attachment, including exposure to ITU, was universally quoted as 2-3 weeks out of a total of 6 years' training. Unfortunately, 30% of anaesthetists in some teaching centres refused students 'access' to anaesthetised patients. Teaching of the compromised airway took place as a formal lecture in 85% of hospitals; with encouragement to attend Accident and Emergency departments, emergency operating theatres and recovery areas emphasised in 55%.

As part of the questionnaire we made a suggestion that 2-3 months of the house year should be spent in a 'resusci-

tation' house job involving supervised periods in anaesthesia, intensive care, coronary care and accident and emergency, familiarising the prospective senior house officer with airway, breathing and circulation problems and their management. This was considered a good idea for all, or at least some, house staff by 5% and 55% of respondents respectively. It was considered a bad idea by 40% of respondents.

We need to do more than comment on the inadequacies of CPR skills. Audit of resuscitation skills is essential. Training must be compulsory, assessed, a component of final examinations and re-assessed throughout a doctor's career. Resuscitation officers should be part of every department and supplied with sufficient time and resources to train and retrain members of the department. If anaesthetists consider the above level of training to be adequate it seems hardly surprising that junior doctors (and by inference senior doctors) of all specialties perform badly in BLS. It is as a consequence of the inadequate level of understanding of the physiology and practice of ABC that our intensive care units continue to accept a stream of patients whose basic management has been substandard causing significant morbidity, mortality and cost.

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Rebreathing and the Humphrey ADE breathing system

It is interesting to read the comments by Dr Humphrey (*Anaesthesia* 1992; **47**: 625) on the Humphrey ADE system. His previous findings [1], that this anaesthetic system is more effective than the conventional Magill during manual ventilation, have since been corroborated by other workers [2]. However, his statements about the greater efficiency of the ADE relative to the Magill attachment during spontaneous ventilation is open to question. The studies [3, 4] reporting on the more favourable fresh gas economics of the ADE system, were all based on the use of minimum inspired carbon dioxide tension ($F_{\text{ICO}_2\text{min}}$) as an indicator of rebreathing. Whilst $F_{\text{ICO}_2\text{min}}$ is easily accessible, given the advent of capnography, many investigators have raised doubts about the reliability of this clinical measurement in detecting the onset of rebreathing [5-8].

Kain and Nunn defined rebreathing to be present when the mixed inspired gas reaching the alveoli contains a concentration of CO_2 greater than could be accounted for by the alveolar gas re-inhaled from the patient's anatomical

deadspace [5]. Consequently, they implied that the measurement of mean inspired CO_2 concentration (with respect to volume inspired) would be appropriate but difficult to apply with semi-closed systems. It would require measurement of inspiratory flows in addition to CO_2 concentration, and an on-line microprocessor which integrates the signals from these analysers similar to that used by Noe [9] and Byrick and Janssen [10]. Kain and Nunn also noticed that the inspired CO_2 is raised above zero during rebreathing, but recognised the flaws of interpreting such changes in relation to the mean CO_2 of the inspired gas. These reservations were reiterated by Conway in his review of breathing systems [11]. Conway commented that rebreathing cannot always be recognised by monitoring inspired CO_2 concentration, for this may be increased during parts of inspiration and decrease to zero during other parts of the inspiratory period. Similar concerns about this measurement point were also voiced by Sykes [6] and Miller [7]. The latter pointed out that a raised minimum inspired CO_2 is a late sign of rebreathing because

of the tendency of plug flow within breathing systems, and consequently only a small volume of unmixed fresh gas need be inspired past the sampling point to yield a $F_{\text{ICO}_2\text{min}}$ value of zero.

Chan and colleagues [12] assessed the sensitivity of this clinical measurement in detecting rebreathing by comparing it with the clinical criteria of Kain and Nunn [5], using a lung model for spontaneous ventilation. These authors demonstrated that the onset of rebreathing was detected significantly later by the $F_{\text{ICO}_2\text{min}}$ method and the results were more variable. Interestingly, the performance of the ADE and Magill systems were similar when the Kain and Nunn criteria were applied. Recently, Soni and colleagues [13] demonstrated significant differences between these two sets of 'rebreathing points' in volunteers using the Magill attachment. In addition to lower fresh gas flows (characterised by a lower fresh gas: baseline minute ventilation ratio, V_F/V_E), rebreathing detected by the $F_{\text{ICO}_2\text{min}}$ method was associated with higher values for inspired volumes of CO_2 and work of breathing. Jennings and colleagues in assessing the enclosed afferent reservoir system (a modified Mapleson A) also predicted a lower V_F/V_E when onset of rebreathing using the $F_{\text{ICO}_2\text{min}}$ definition was applied [14].

Miller [7] avoided the ambiguities associated with the definitions of rebreathing and defined rebreathing as the point at which functional or airway deadspace starts to increase. He used this measurement point to evaluate the performance of breathing systems during spontaneous ventilation. His study emphasised the functional similarity of the A systems tested, including the ADE system. The determination of the onset of rebreathing using this method [15] also appears to be more sensitive than the clinical criteria of Kain and Nunn. The similarity of the current Mapleson A systems, in terms of fresh gas flow requirements, have also been predicted using the graphical model of Dorrington and Lehane [16].

Thus, when more sensitive criteria are used, the ADE system appears to have comparable performance to other Mapleson A systems during spontaneous ventilation. Surely it is high time that when claims of fresh gas economics on breathing systems are made, they should be qualified precisely and acknowledgment made of the current mass of evidence questioning the sensitivity of $F_{\text{ICO}_2\text{min}}$ to detect the onset of rebreathing.

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The 'Whoosh' test in children

It is with considerable interest that we read about the 'Whoosh' test (*Anaesthesia* 1992; 47: 57-8).

In paediatric practice, the use of caudal anaesthesia and analgesia can prove to be a useful adjunct to urological, rectal, and orthopaedic surgery. The caudal approach to the epidural space may offer a greater degree of convenience and a reduced incidence of dural puncture and associated complications. On this basis, a simple and reliable test with a zero false-positive rate would offer considerable advantage for paediatric anaesthesia, as the same problems of inaccuracy of needle placement do occur.

However, we wish to report significant difficulty in interpreting this test in two children. Using a 23 gauge needle through the sacrococcygeal membrane, the correct placement was tested using our standard technique of palpation of the overlying skin during an air injection of 0.5 ml for subcutaneous emphysema and crepitus, and the

'Whoosh' test. In both cases the needle was placed in the epidural space as elicited by the subcutaneous air on injection, and high resistance to injection of normal saline. However, using auscultation over the thorax revealed a 'whoosh.' This was not the crackling sound suggested by the authors, but a pitched clearly heard rumble. When corrected whoosh test revealed the same result as the slightly reduced volume.

Therefore, in these children (both around 10 years old) it is impossible to differentiate between correct and incorrect placement on the basis of the 'whoosh' test. The response from the incorrectly positioned needle was more positive than the correctly placed one.

While the 'whoosh' test may have a role in confirming correct placement, we feel that the two false-positive

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test is not reliable in paediatric practice. It is likely that the shorter distance between the thoraco-sacral spine and the highly compliant tissues of small children aid in the transmission of the noise generated by the injection of air.

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When should blood pressure be measured before anaesthesia?

Measurement of arterial blood pressure before anaesthesia is important for three reasons: firstly, to identify patients with a diastolic pressure greater than 110 mmHg, who would benefit from postponement of elective surgery until their blood pressure is controlled [1]. This is because they are at risk of exaggerated pressor responses to laryngoscopy and tracheal intubation, and they may have labile blood pressure during anaesthesia. Secondly, blood pressure is measured before anaesthesia to identify those patients with diastolic pressures between 90 and 110 mmHg. These patients are at risk of similar problems although to a lesser extent, and the anaesthetist may decide to proceed with anaesthesia with extra caution. Thirdly, blood pressure is measured to establish a baseline for anaesthetic management.

Colquhoun and Rabey [2] have shown that a diastolic pressure of (or greater than) 90 mmHg measured by the ward nurse is not a good predictor of increased diastolic pressure in the anaesthetic room. Other workers have also suggested that more than one blood pressure measurement should be made before anaesthesia. For example, Vertes [3] suggests that three blood pressure measurements should be made before operation, so that blood pressure lability as well as the absolute value can be assessed.

We report a small, closely controlled study, which was carried out to determine whether the admission blood pressure measured by the ward nurse is relevant to the premedicated patients that we anaesthetise. Fourteen consecutive patients, all scheduled for transurethral resection of the prostate by the same surgeon, and all anaesthetised by the same anaesthetist were studied. Three blood pressure measurements were recorded. The first was the lowest blood pressure recorded by the ward nurse before operation. The second was the blood pressure recorded by the anaesthetist at the pre-operative visit, and the third was a pre-induction measurement made by the same anaesthetist in the anaesthetic room. All measurements were made using the Riva-Rocci method; a mercury sphygmomanometer was used by the ward nurse and an aneroid one, (previously calibrated against a mercury sphygmomanometer) was used by the anaesthetist. All the patients were premedicated with intramuscular papaveretum and hyoscine. The dose was calculated from ASA status and age, using a system in which the dose decreased with increasing ASA status and age. The times of premedication and anaesthetic room blood pressure measurement were recorded. Patient information and the

differences in blood pressure were compared using Student's paired *t*-tests.

The mean differences in blood pressure in mmHg, are shown in Table 1. No statistically significant difference was found between the systolic, diastolic and mean pressures measured by the ward nurse and those measured by the anaesthetists in the anaesthetic room. Similarly there was no significant difference between the pressures measured by the ward nurse and those measured by the anaesthetist at the pre-operative visit. However, the systolic, diastolic and mean pressures measured by the anaesthetist on the ward were all significantly higher than those measured in the anaesthetic room. No relationship was seen between the dose of premedication, or the time since premedication, and the difference in blood pressure measured on the ward and in the anaesthetic room.

This study shows that the lowest blood pressure measured by the ward nurse before operation gives a good prediction of blood pressures in the anaesthetic room. No such prediction is provided by the blood pressure measured by the anaesthetist at the pre-operative visit. The fact that the pressures measured by the anaesthetist on the ward were higher than those in the anaesthetic room may be because of the phenomenon of 'white coat' hypertension in the unpremedicated patients on the ward [4]. Alternatively, it could be due to the hypotensive effect of premedication, although this is not supported by the lack of correlation with time and dose of premedication. The blood pressures measured on the ward by the anaesthetist tended to be higher than those measured by the nurse, but the difference did not reach statistical significance. Although this is a small study, it was well controlled since all the patients were male, and they all had the same premedication and the same operation. Our results show that the lowest blood pressure recorded by the ward nurse before operation, in this and possibly other groups of patients, can be used not only to identify at-risk patients, but also as a baseline for anaesthetic management.

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Table 1. Mean differences in blood pressure in mmHg (SD) between the three groups.

	Ward nurse: anaesthetist in anaesthetic room	Anaesthetist on ward: anaesthetic room	Ward nurse: anaesthetist on ward
Systolic	+3.00 (22.87)	+11.57 (12.38)**	-8.57 (20.28)
Diastolic	+6.29 (13.21)	+5.29 (7.34)*	-1.00 (15.20)
Mean	+6.00 (14.76)	+7.36 (7.65)**	-1.36 (14.66)

*P < 0.05. **P < 0.005.

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Arterial hypoxaemia and intrapulmonary shunt

The way in which a reduction in cardiac output will cause arterial hypoxaemia if an intrapulmonary shunt is present was detailed by Dr Westbrook and Professor Sykes (*Anaesthesia* 1992; **47**: 307–10) using the Macpuf computer model, and again described by Professor Sykes in his review of clinical measurement (*Anaesthesia* 1992; **47**: 425–32). More contentious is the way in which shunt fraction ($\dot{Q}_s/\dot{Q}_t$) changes with cardiac output. They suggest that an inverse relationship exists because of the likely accompanying changes in hydrostatic pressures, and point out that this would augment the fall in oxygen saturation (Sao_2) seen after a decrease in cardiac output.

We know of little evidence that this inverse relationship holds in man. Many animal experiments, particularly in dogs after intravenous administration of oleic acid which produces an oedematous lung injury characterised by intrapulmonary shunting, suggest that the reverse is true, i.e. that $\dot{Q}_s/\dot{Q}_t$ varies directly with cardiac output [1], and that this is a result of the associated change in mixed venous Po_2 modifying hypoxic pulmonary vasoconstriction [2]. It seems probable that, despite the different pathology, such a mechanism would also apply in anaesthetised man, in which case changes in Sao_2 resulting from changes in cardiac output would be minimised.

The situation is complex, but we have given one reason why in our clinical practice (unlike the experience of the Oxford authors) we are struck by how frequently a decrease in cardiac output, enough to cause a substantial transient reduction in end-tidal Pco_2 in a patient whose lungs are being ventilated, leaves the Sao_2 unaffected.

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A reply

We thank Drs J.M. Handel and N.J.H. Davies for their comments on our paper. We programmed the Macpuf computer model with a fixed shunt and then observed the

effects of decreasing cardiac output at each level of shunt. The resulting decreases in saturation are, therefore, due entirely to decreases in the saturation of the mixed venous blood perfusing the shunt.

We agree that the second mechanism postulated for the decrease in saturation, a decrease in flow to the oxygenated nondependent areas of lung resulting in an increased percentage of shunt, has not been demonstrated in man. However, it has been proposed as an explanation for the increase in percentage shunt observed when pulmonary artery pressure is reduced by haemorrhage in dogs with a dependent collapsed lobe [1]. Since the compression collapse observed by Hedenstierna's group during anaesthesia [2] is invariably in the dependent zones, there is no reason why the same mechanism should not occur in man. As we stated in our paper, the object was to make anaesthetists aware of these two possible mechanisms in the hope that it would stimulate further study.

We acknowledge that there have been many publications showing a direct relationship between cardiac output and shunt, and that the changes may be due to the effects of mixed venous Po_2 on hypoxic pulmonary vasoconstriction. However, as we pointed out, there are many factors which decrease the effectiveness of this mechanism in the dependent zones of the lung in the closed chest situation and the model of pulmonary oedema used by Sandoval *et al.* [3] is of doubtful relevance to the conditions existing in the anaesthetised patient.

The failure of Drs Handel and Davies to observe arterial desaturation during blood loss may reflect a difference in their clinical practice. Our patients were undergoing large bowel surgery, were usually dehydrated by pre-operative bowel preparation and were often subjected to intra-operative ultrasound examination of the liver which probably increased dependent zone collapse.

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Laryngeal mask and bleeding diathesis

I would like to make some comments about the recent correspondence on the use of the laryngeal mask airway (LMA) in the presence of a bleeding diathesis (*Anaesthesia* 1992; **47**: 530–1 and 531–2).

Thompsett and Cundy report a laryngeal haematoma developing after an 'easy' LMA insertion in a patient with a severe myelodysplastic syndrome. The patient required

exploration of a persistent bleed caused 2 days previously by insertion of a subclavian line. The haematoma presented as stridor during the operation and was diagnosed at direct laryngoscopy after the LMA was removed. The patients' trachea was subsequently intubated and he died 4 days later. Commenting on this letter, Brain suggests that the haematoma could have occurred secondary to subclavian

line insertion itself rather than insertion of the LMA. He also points out that laryngeal trauma may be avoided by use of an appropriate insertion technique and goes on to suggest how misplacement of the tip can be diagnosed.

I was surprised that neither letter made mention of the potential of fiberoptic laryngoscopy in the diagnosis and management of these problems. Partial airway obstruction, such as occurred at the beginning of this case, can be rapidly and noninvasively assessed by passing a fiberoptic instrument down the LMA to the level of the mask aperture bars. Similarly, it can help diagnose misplacement of the LMA, allowing it to be repositioned if the tip is lying in the laryngeal vestibule.

The management of the airway in the presence of a bleeding disorder is always potentially hazardous. In circumstances where a facemask cannot be used, the LMA may be equally appropriate to a tracheal tube, or even a

facemask plus oral airway, if airway maintenance is difficult. I would like to suggest that the presence of a bleeding disorder is only a relative contraindication to LMA insertion. When the LMA is used, it may be possible to reduce the risk of bleeding by inserting it slowly using the standard approach. Fiberoptic laryngoscopy, performed at insertion and regularly thereafter, will allow the early detection of problems.

Finally, this case serves to highlight the hazards of using the subclavian route for central line insertion in a patient with a bleeding diathesis. If the jugular veins had been used, persistent bleeding might have been simply managed with external pressure rather than general anaesthesia and surgical exploration.

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Laryngeal mask airway and radiotherapy in the prone position

I read with interest the correspondence by Dr N. Kee (*Anaesthesia* 1992; 47: 446-7) on the use of the laryngeal mask airway (LMA) for radiotherapy in the prone position. There is no doubt that the LMA has simplified anaesthesia for radiotherapeutic and radiodiagnostic procedures in children and contributed greatly to the safety of these procedures where maintenance of the airway patency is made more difficult by the positions required during radiotherapy. The LMA eliminates the need for laryngoscopy and avoids the possible morbidity of frequent tracheal intubation. However, the LMA affords no protection against pulmonary aspiration. Dr Kee describes one episode of '... minor regurgitation at induction' and then proceeds with general anaesthesia in the prone position. To continue using a LMA, at least on this occasion, might be construed as hazardous.

Rowbottom and Simpson [1], using fiberoptic laryngoscopy, have shown that partial respiratory

obstruction occurred in up to 50% of children when an LMA was used; this was difficult to detect clinically. As a result, gastric distension could occur if assisted ventilation were used, and combined with the increase in intra-abdominal pressure caused by the prone position would increase the risk of regurgitation and pulmonary aspiration.

The concept of using the LMA for radiotherapy seems simple, but is far from being totally safe, and anaesthetists should be careful in using this technique for the prone position.

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The laryngeal mask airway—suboptimal availability, a cause for concern

The laryngeal mask airway (LMA) has revolutionised anaesthetic practice. Increasing numbers of reports of its use under various circumstances are appearing in the literature. Having worked at numerous hospitals, it is our impression that the LMA is not as freely available as it should be: it may not be available in the Accident and Emergency or X ray departments, or indeed in certain anaesthetic rooms in the main operating theatres. Not infrequently, the ODA has to borrow one from an adjacent operating theatre, and occasionally one has to go without. We are all aware of the scenario of a failed tracheal intubation in a patient in whom oxygenation cannot be maintained by facemask. Recently one of us (A.R.W.) was unable to intubate the trachea or maintain adequate

oxygenation via a face mask during a Caesarean section, but fortunately an LMA provided an excellent airway and the operation was completed with the patient breathing spontaneously. Had this not been available the consequences might have been catastrophic.

Disasters due to loss of airway control are, fortunately, uncommon. However, is it not time that it becomes departmental policy that LMAs should be present, in suitable sizes, on all intubation trolleys throughout the hospital?

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A.R. WILLIAMS
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Combined spinal-extradural anaesthesia for Caesarean section

We read with interest the article by Lyons (*Anaesthesia* 1992; 47: 199-201). Unlike the authors, we strongly recommend the use of single space combined spinal and

extradural anaesthesia (CSE) as the regional anaesthetic technique of choice for Caesarean section. Any regional technique that reduces the requirement for general

anaesthesia has to be recommended and none of the patients in the single space group in the study mentioned required conversion to general anaesthesia. Failure rates ranging from as low as 0.46% to as high as 35% have been quoted for subarachnoid anaesthesia [1]. We were disappointed to see a failure rate of 16% for subarachnoid anaesthesia in the single space group. This, we feel can be reduced to more acceptable levels by greater attention to technical detail, namely by using not only needles of appropriate size and resistance, but also by waiting for cerebrospinal fluid (CSF) to appear at the hub of the needle prior to injecting. When using a single space technique, Randalls *et al.* [2] reported a failure rate of 4%, with a 26 gauge Quincke point needle, while Carrie and Donald, using a 26 gauge pencil point needle, reported no failures [3].

We have recently finished conducting a study which compared the resistance to fluid flow of the 26 gauge, 120 mm long Braun spinal needle with that of the 27 gauge, 120 mm long Becton Dickinson (BD) needle. This was done by using a pressure infusion monitor pump (IVAC 560), a technique previously described by Morris *et al.* [4]. Surprisingly, we found that the mean resistance to fluid flow for the 26 gauge needle was twice the mean resistance for the 27 gauge needle. The findings of this experiment were confirmed by a controlled randomised study in 100 obstetric patients who required regional anaesthesia. We found that the time taken from dural puncture to appearance of CSF was on average about three times faster with the 27 gauge needle. The failure rate for subarachnoid anaesthesia with the 27 gauge needle was 4% and with the 26 gauge needle was 10%. These failure rates are lower than the 16% reported by Lyons and his colleagues.

We feel that needles of low resistance can greatly increase the success rate of subarachnoid anaesthesia for a number of reasons. Firstly, the time taken for CSF to appear at the hub of the needle is decreased and secondly, not only is aspiration of CSF during and after injection much easier, but also loss of local anaesthetic during injection is less likely. Finally, they are less likely to suffer from blockage and the ability to inject quickly and easily reduces the incidence of patchy blocks. In fact, some of the problems associated with needles of high resistance have already been highlighted by the authors in their evaluation of the 30 gauge needle for single space CSE technique [5]. Since the completion of the above study we have used the 27 gauge needle in 190 patients with a failure rate of only 3%.

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We were interested to read the account of combined epidural/spinal anaesthesia for Caesarean section from Drs Lyons, Macdonald and Mikl (*Anaesthesia* 1992; **47**: 199–201). We wish to make two observations.

When discussing the 'double space technique' employing separate epidural and spinal punctures, the authors do not make clear which procedure is performed first. A recent experience in this unit has led us to advise performing the epidural puncture *before* dural puncture. A large woman was scheduled for elective Caesarean section, and had requested regional anaesthesia. A combined technique using separate punctures had been explained and agreed. Dural puncture was achieved uneventfully at the L₃₋₄ interspace by an experienced senior registrar, with the woman sitting up. After injection of heavy bupivacaine, several attempts were made to site the epidural at L₂₋₃. This proved extremely difficult, and after achieving a bloody tap with the epidural catheter, assistance was sought from the consultant obstetric anaesthetist. The epidural space was cannulated via the L₃₋₄ interspace. Despite appropriate positioning, the level of spinal anaesthesia remained at T₁₀ and it proved impossible to extend it above T₇ with the epidural. The woman was offered a second spinal but declined, and was given a general anaesthetic.

Although siting the epidural catheter before performing spinal block carries a theoretical risk of transfixing the epidural catheter with the spinal needle, we have been unable to do this *in vitro* when using 24 gauge Sprotte or 25 gauge Whitacre tipped spinal needles. Once the epidural catheter has been successfully inserted dural puncture can proceed in an unhurried manner, rendering the technique suitable for teaching juniors.

We are surprised at the failure rate of the 'through needle' technique reported. Since this technique was introduced into our maternity unit 4 months ago we have performed 78 Caesarean sections (emergency and elective) using Tuohy guided spinal anaesthesia with a 119 mm 25 g Whitacre needle. In only one case was dural puncture impossible, and in three women the epidural was used to extend the level of spinal anaesthesia *before* starting surgery. This represents a failure rate of 5% using Dr Lyons' criteria. None of the junior anaesthetists had previous experience of the technique, but it has been accepted enthusiastically. Neither we nor our patients would choose to return to the technique of separate spinal and epidural punctures.

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A reply

Thank you for the opportunity to reply to Drs Roberts and Brighouse. We agree with their comments on order of procedure.

At the time of writing we have performed 956 Caesarean sections using combined spinal/epidural anaesthesia and twice have attempted to introduce the 'through the Tuohy' technique. In our hands separate punctures work best. The point to emphasise is, that whichever of these techniques is used, failure of regional anaesthesia, and recourse to general anaesthesia, is a very rare event.

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Spinal anaesthesia for Caesarean section in an achondroplastic dwarf

A 26-year-old primigravid achondroplastic dwarf was to be delivered by elective Caesarean Section at 36 weeks gestation. At 21 weeks gestation she had suffered a deep venous thrombosis and pulmonary embolism, and was treated initially with intravenous heparin followed by warfarin. At 33 weeks gestation, she was re-admitted to the maternity unit and the warfarin was substituted by intravenous heparin to continue until delivery. The pregnancy was further complicated by polyhydramnios. Examination revealed the typical features of achondroplasia. She weighed 44 kg, and was 117 cm tall with a relatively normal spinal length of 52 cm. Subarachnoid block using a microspinal catheter was chosen to provide anaesthesia for the section.

Heparin was stopped 24 h before surgery, and a clotting screen was normal on the day. Oral ranitidine 150 mg was given the night before, and on the morning of surgery. A circulatory preload of 6 ml.kg⁻¹ of Ringer lactate solution was given. With the patient in the left lateral position, a 32 gauge catheter was passed 4 cm into the subarachnoid space via a 25 gauge needle at the L₂₋₃ interspace. An initial infusion of 0.5 ml hyperbaric 0.5% plain bupivacaine was given through the catheter. Within 20 min, a bilateral T₆ block had developed. Blood pressure decreased to 60/34 mmHg and was treated with 12 mg intravenous ephedrine. There was a delay in starting surgery during which the block receded over a 15 min period to T₈ on the left and T₁₀ on the right. A further 0.5 ml local anaesthetic was given which restored the block

to T₄ bilaterally. Surgery was begun with the patient in the wedged supine position.

A lower uterine segment Caesarean section was performed, and a live, female achondroplastic child, with good Apgar scores, was delivered 14 min after skin incision. Forty minutes after the second dose of bupivacaine, a further 0.3 ml was given. A total of 0.1 mg diamorphine was given via the catheter to provide postoperative analgesia. The motor and sensory blocks wore off within 30 min of the end of the procedure. The diamorphine provided excellent analgesia in the first 24 h. Mother and baby were discharged after 7 days.

There are few reports describing the use of central blockade in the anaesthetic management of achondroplastic patients [1, 2]. If spinal anaesthesia is to be undertaken, we would recommend the use of a microspinal catheter, since this allows small incremental doses of local anaesthetic to be given when necessary.

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Cerebrospinal fluid—a reliable guide to dural puncture?

I have read with interest the recent correspondence about the complications and management of post-accidental dural puncture in mothers. I was surprised that no reference was made to the difficulties in identifying the epidural space after an inadvertent dural puncture has occurred. The following case report illustrates some of these difficulties.

Following an inadvertent dural puncture in a 32-year-old primigravida, confusion arose when further attempts were made to identify the epidural space. The second epidural was inserted immediately after the dural puncture had occurred, the space being located using a Tuohy needle and loss of resistance to air. Before the catheter was inserted cerebrospinal fluid (CSF) appeared through the needle. This was thought to be due to the leak from the previous puncture and the local anaesthetic was injected once the catheter was introduced. A total dose of 8 ml bupivacaine was given in increments and a unilateral block was produced which could not be rectified easily. Further confusion arose when a spinal injection for Caesarean section became necessary, as the patient expressed a desire to remain awake. This was attempted with a 26 gauge needle and CSF aspiration was taken as an indication of the subarachnoid placement of the needle; 2.8 ml of bupivacaine was injected which caused patchy anaesthesia restricted to below the L₅ dermatome.

The third epidural injection was 48 h post-delivery for the relief of postdural puncture headache. Again, the epidural space was clearly identified with lack of resistance to air, but 60s later clear fluid appeared through the needle. However, as there was little doubt that the needle was in the epidural space, 20 ml of blood drawn apically from the patient was injected with remarkable improvement in her headache.

Confusion in identifying the epidural space after an inadvertent dural puncture has serious implications. If only a dry tap is accepted as an indication of entry in the epidural space, then some patients may be subjected to further unnecessary epidural injections with increased risk of dural puncture. The presence of CSF in the epidural space may make subsequent identification of the subarachnoid space difficult if spinal anaesthesia is contemplated. It is proposed that this was the cause of the failed spinal anaesthesia in the case reported. If a blood patch is to be used, the subarachnoid injection of 20 ml of blood is potentially hazardous [1].

It is very difficult to suggest a test that will distinguish CSF obtained from the epidural as opposed to the subarachnoid space. The rate of flow of CSF from a Tuohy needle is not an adequate guide to the correct placement of the needle as was shown by Hollway and Telford [2]. The experience of the operator is probably the best asset in coping with these potentially confusing situations.

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Alfentanil and raised intracranial pressure

We read with interest the report by Dr Moss (*Anaesthesia* 1992; 47: 134–136) concerning increases in intracranial pressure associated with the use of alfentanil in patients with normal pressure hydrocephalus and wish to describe a similar event in a child with a head injury.

A 30 kg, 8-year-old boy suffered a head injury while riding his bicycle. His trachea was intubated and his lungs ventilated. A CT scan showed a left parietal contusion, a small left subdural haematoma with a slight mass effect and a small amount of subarachnoid blood. A subdural catheter was inserted through a frontal burr hole and the child transported to the intensive care unit for sedation, ventilation and intracranial pressure (ICP) monitoring. Infusions of morphine and midazolam were used for sedation and atracurium for neuromuscular blockade. His lungs were hyperventilated to a P_{aCO_2} of 3.5 to 4.0 kPa. ICP remained steady at 10 to 12 mmHg. Several hours later, during a nursing procedure, his pulse and blood pressure rose, although ICP remained steady. His level of sedation was considered inadequate and therefore a 0.5 mg bolus of alfentanil was given to provide a short period of increased sedation. Immediately following this, the ICP showed a brisk rise from 10 to 27 mmHg which lasted for 20 min. Mean arterial pressure (MAP) fell from 85 to 75 mmHg and pulse rate from 110 to 90 beat.min.⁻¹. This resulted in a decrease of cerebral perfusion pressure from 75 to 47 mmHg. Two hours later, a rise of ICP from 10 to 30 mmHg followed a further bolus of 0.5 mg alfentanil, which required treatment with mannitol. Following this, fentanyl was substituted for alfentanil. Two doses of 50 µg were used for sedation; these had no effect on ICP despite evidence of an overall steady decrease in intracranial compliance.

As Dr Moss reports, the effects of alfentanil on cerebral vasculature are not clear. Marx *et al.* [1] described an increase in cerebrospinal fluid pressure associated with the use of alfentanil, which also caused a significant decrease in MAP. Fentanyl did not exhibit this action. It was unclear whether the increase in ICP was due to a direct cerebral vasodilator effect or secondary to the decrease in MAP. Cuillerier and colleagues [2], however, did not show any difference between alfentanil, fentanyl and sufentanil in ICP or cerebral perfusion pressure.

In two of Dr Moss's patients, the increase in ICP was not associated with a significant reduction in MAP, and preceded it. It seems more likely, therefore, that the rise in ICP is due to a direct effect on cerebral vasculature rather than a compensatory response to a decrease in MAP. Like Dr Moss, we would also advise caution with the use of alfentanil when intracranial compliance is compromised.

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Patient-controlled analgesia—a serious incident

Drs Grover and Heath (*Anaesthesia* 1992; 47: 402–4) mention their use of a Vygon 'one-way valve' to prevent reflux of opioid into the gravity feed intravenous infusion. However, it appears that they did not use an 'antisiphon' valve to prevent the syphoning of opioid out of the syringe in the PCA pump. This would have prevented the incident that they describe. 'Y' connector sets that incorporate both antireflux and antisiphon valves are available, cheap and have been standard practice in our hospital for several years. The incident brings to six the number of known episodes of malfunction of a PCA pump leading to serious morbidity or mortality [1], but must be seen against the extensive worldwide use of the technique, the fact that all equipment will malfunction at some time, and that all routes of opioid administration carry risks that are largely undetermined [2]. It is significant that five out of the six patients survived.

The authors go on to discuss the problems of monitoring of patients, commenting on previous work from this hospital [3]. They state 'it is not surprising if busy nurses accord a low priority to quantifying an event which apparently provokes little response from the medical team'. Here we always recognise that respiratory rate is a poor indicator of respiratory function, (except when very low or very high). However, at present there is no other simple measure of monitoring respiratory activity that can be used easily on ordinary wards. Nurses in this hospital always have a positive response from anaesthetists on reporting possible respiratory problems, even if no active

intervention is needed. Monitoring of respiration is deemed mandatory and not an option that could be given a 'priority'.

From time to time we see failure to monitor or failure to act on behalf of patients who have clearly deranged physiological parameters. Our nursing colleagues must take up the issue of the provision of a safe environment for patients who are recovering from major surgery. Surgeons and anaesthetists are now auditing their morbidity and mortality. Nurses too must take on this type of audit and show that if they change their practice (e.g. team nursing) and move away from the traditional graded and progressive care (i.e. the illest patients in one area) then they must demonstrate that there is an improvement in safety and a reduction in morbidity.

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Patient-controlled analgesia in burn patients

Drs Choiniere, Grenier and Paquette demonstrate the benefits of patient-controlled analgesia in burns (*Anaesthesia* 1992; **47**: 467-472). They conclude 'that PCA is a safe, effective and improved method of controlling burn pain'. Whilst they demonstrate adequately the latter two points, there is no evidence for the former from their study

based on 12 patients. I share the same opinion with them that PCA is safe, but much larger studies will be necessary to demonstrate this conclusively.

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Fatal theophylline poisoning with rhabdomyolysis

In their report of a patient with severe theophylline intoxication, Drs Parr and Willatts (*Anaesthesia* 1991; **46**: 557-9) draw attention to important aspects of the management of such cases. Nevertheless, questions should be raised about this particular case which are not discussed by the authors.

It was evident within a short time of initial presentation that the patient had ingested a substantial overdose of a drug well known to cause life-threatening complications [1, 2]. Despite this, the patient was transferred from the Accident and Emergency Department to the Coronary Care Unit (CCU) and only gained admission to the Intensive Therapy Unit (ITU) several hours later after suffering a cardiorespiratory arrest. The convulsions and cardiac arrhythmias which preceded the final transfer were probably predictable, given the magnitude of the overdose and the dangerously high theophylline level. Cardiorespiratory collapse may have been avoided, and its complications prevented, if the patient had been admitted directly to the ITU. After admission to ITU, the treatment received by the patient appears to have been thorough and appropriate; however, there are indications that the management in the CCU was illogical and may have contributed to the patient's deterioration.

For instance, supplementary oxygen therapy was apparently not given and, consequently, the initial blood gas analysis showed significant hypoxaemia. Potassium was administered in an inadequate dosage considering the admission serum potassium concentration of 2.4 mmol.l⁻¹. Furthermore, following unsuccessful therapy with a single small dose of amiodarone, disopyramide was used. Theophylline acts by inhibiting phosphodiesterases and therefore increases cyclic AMP levels [3], the arrhythmias and hypotension seen in theophylline overdose are attributable to this effect. Treatment of theophylline-induced arrhythmias is probably best undertaken using a beta-adrenoceptor antagonist such as propanolol [4, 5] which prevents the conversion of ATP to cyclic AMP, rather than a drug such as disopyramide which has strong anticholinergic effects and is probably inappropriate in circumstances of increased adrenergic activity. Given this mode of action, the rapid development of ventricular fibrillation following the administration of disopyramide might have been predicted. This sequence of events raises the question of whether the CCU is ever an appropriate place to monitor and manage patients who have taken potentially dangerous overdoses.

Although survival has been reported after higher blood theophylline levels [6, 7] recovery could not be guaranteed in the case described. However, it is essential that prompt recognition and treatment of a deteriorating clinical state occurs if the chances of survival are to be enhanced. This requires continuous monitoring and prompt intervention by an immediately available and suitably experienced doctor at all times. For this reason, patients who are

known to be significantly intoxicated with potentially dangerous drugs should be directly admitted to the ITU for observation. This case history highlights the need for improved training of junior medical staff and greater consultant involvement in the acute specialities.

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A reply

Thank you for the opportunity to comment on the letter from Drs Taylor, Smith and McQuillan which discusses our case report on fatal theophylline intoxication. We agree whole-heartedly with their views. The most important consideration, however, is how can such a sequence of events be avoided in the future?

Training in intensive therapy should be available to all medical students and doctors training in acute medical specialties. The purpose of this is to enable them to recognise acute illness or deterioration in their patient's condition at a time when rapid admission or transfer to an Intensive Therapy Unit can benefit the patient. It is with great pleasure that we can confirm the initiative of the Royal Colleges of Anaesthetists, Physicians and Surgeons which outlines a common core of training in intensive therapy suitable for trainees from all specialties.

In due course this will overcome such problems as misplaced admission to a Coronary Care Unit, as in this case, and will result in better patient care.

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Pulse oximetry in pulseless patients

We read with interest the case reported by Dawalibi *et al.* (*Anaesthesia* 1991; 46: 990–1) in which an oximeter (Ohmeda Biox 3700) continued to provide SpO_2 and heart rate readings from an organ donor for 15 min following cardiectomy. While we agree with the authors that pulse oximeter readings should not be accepted uncritically, we wish to report our experience of a similar case in which apparently reliable readings were obtained.

Saturation was measured using a Nellcor N-200-E pulse oximeter (oximeter software version 2.9, powerbase version 2.7). Data from the oximeter communication port were stored on computer using methodology we have previously described [1], so we are able to reproduce the record here (Fig. 1). Prior to cardiectomy, episodes of desaturation were recorded (0.4–0.6 h) which were associated with pulmonary oedema. Following this, SpO_2 was well maintained until cardiectomy. During cardiectomy, SpO_2 rapidly declined to around 85%, after which the oximeter displayed a saturation of zero and its pulse-search warning was illuminated. At this time there was a change in the monitor status of the oximeter from 11 (indicating pulse search status: locked) to 10 (indicating pulse search status: search) which coincided with the recorded time of clamp placement and cessation of ventilation. While no independent estimates of SpO_2 were obtained, all values provided by the oximeter are plausible in the light of the donor's condition.

A possible explanation for the discrepancy between these results and those of Dawalibi *et al.* is use of a different type of oximeter. Our observation suggests that limitations of oximeters may differ from one brand to another. This is

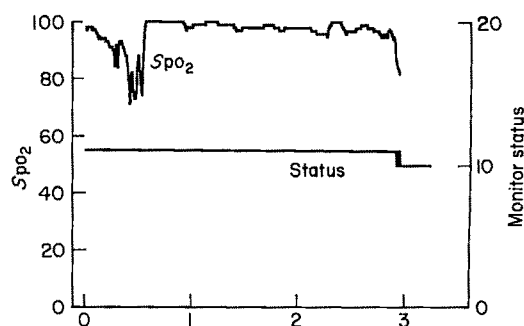


Fig. 1.

consistent with the fact that different brands of oximeters use different algorithms [2] in the calculation of SpO_2 .

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Intra-aortic balloon pumps and the pulse oximeter

The pulse oximeter has become an indispensable tool in operating theatres and intensive care units, because it provides an immediate and continuous measure of oxygen saturation. Many potential errors using pulse oximetry have been elucidated [1]. I present here another such error with an easy solution.

A 58-year-old man required an intra-aortic balloon pump following aortic root replacement with coronary artery insertion. The abnormal pulse waveforms prevented correct functioning of the pulse oximeter. The radial arterial waveform had a triple peak, whilst distal to the balloon pump there was a double peak, which was palpable in the dorsalis pedis arteries. The three peaks of radial artery pressure ranged from 100 to 85 mmHg with a diastolic value of 60 mmHg and a heart rate of 120 $\text{beat}\cdot\text{min}^{-1}$. The pulse amplitude display of the Nellcor 100E pulse oximeter showed these double and triple peaks, but it was unable to 'lock on' to them to produce any display of heart rate or saturation and there was no pulse tone. Neither pauses nor ratio changes in balloon pumping (being tried by the surgeon), nor changing the site of the probe or the oximeter averaging time, solved the problem.

To overcome the problem the radial pulse was occluded by external finger pressure sufficient to remove any signal display on the oximeter. Intermittent release of this pressure in phase with the first peak of the pulse was performed using the rhythmic sound of the balloon pump and the arterial trace for timing. This produced a single pulse at the pulse oximeter probe and within a few seconds the audible pulse tone sounded, and saturation and heart rates corresponding to the ECG rate were displayed. Once 'locked on' the oximeter continued to correctly display heart rate once the external pressure was completely released. This was repeated three times to confirm the

findings, making sure to switch off the oximeter completely before each attempt. It was then performed twice on the great toe again overcoming the problem. The calculated saturation from an arterial sample was 97% and compared well with the oximeter value of 98%.

In order to show that these findings were reproducible, a second patient with an intra-aortic balloon pump was studied. There was a double pulse at the radial artery and a single pulse at the toe. The radial artery pressure was 145/80 mmHg with a heart rate of 115 $\text{beat}\cdot\text{min}^{-1}$. The single pulse in the foot was identified by the oximeter as a rate of 115 $\text{beat}\cdot\text{min}^{-1}$ and saturation of 98%. The double pulse of the radial artery showed on the pulse amplitude display of the oximeter, with a saturation of 97% but a very unstable rate varying from 210 to 230 $\text{beat}\cdot\text{min}^{-1}$ (about twice that of the heart rate). Arterial occlusion with intermittent release, as above, produced a saturation of 96% with a rate of 113 $\text{beat}\cdot\text{min}^{-1}$ and this was maintained following release.

The pulse oximeter calculates saturation from the pulsatile component of the red and infra-red absorption, and a pulse rate which correlates with the ECG heart rate is a useful way of validating the readings. It is important to allow a pause of about 20 s between releasing the occlusion and noting the saturation, in order to exclude any interference from venous pulsation caused by the intermittent occlusion of the pulse. It is also important to confirm with intra-arterial monitoring that the double or triple pulses shown on the oximeter are all arterial, as venous pulsations can cause erroneous saturations [2].

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Obstruction due to wet soda lime granules

The circle system with CO₂ absorption has increased in popularity due, presumably, to the desirability of using expensive inhalational agents more economically, coupled with the increased availability of gas and vapour analysis. As an exercise in frugality, as soon as the concentrations of oxygen and nitrous oxide in the circle have reached approximately 30%:70%, I reduce the fresh gas flow to 300 ml.min⁻¹ of each gas and make minimal adjustments as necessary. Unfortunately, after an hour or so, the manometer dial shows increasing PEEP, up to 8 cmH₂O. This is due to resistance to gas flow through the soda lime (in a two-tiered canister with a total volume of 3l). The resistance is caused mainly by condensation of water in the tier which is not absorbing CO₂ and is therefore cold. On inspection, the granules are found to be caked together and partly turned to slurry.

Interference with expiration caused in this way will result in a rise in mean intrathoracic pressure, which, by inhibiting venous return, could reduce cardiac output if the patient is hypovolaemic or vasodilated. The increased

engorgement of veins could be expected to increase bleeding in some circumstances. In cardiac and thoracic surgery, the increased distension of the lungs may impair operating conditions. In craniotomy, unwanted PEEP can cause bulging of the brain. The problem could be avoided by using a (wastefully!) generous fresh gas flow from the Boyle's machine, which will promote the elimination of much more gas plus water vapour through the spill valve, and thus prevent the cold granules from becoming waterlogged. Alternatively, fill one tier (1.5l) with soda lime, which becomes warm and stays dry, while leaving the other tier empty. Copious beads of water condense on the wall of this empty, cold chamber, causing no obstruction to gas flow, and there is no PEEP. It is thus possible to economise on fresh gas flow without performing an unwanted Valsalva manoeuvre.

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P.S. COSSHAM

Nil by mouth after midnight

I was interested to read Drs Dunnet and Zorab's letter (*Anaesthesia* 1992; **47**: 638) regarding removal of dentures and pre-anaesthetic fasting. In July 1991 we too changed our policy and now allow wearers of partial or full dentures to keep their teeth in until arriving in the anaesthetic room.

At the same time we relaxed our guidelines on pre-operative fluid restriction and now allow patients to receive 50 ml of clear fluid (water or squash) hourly until 2 h pre-operatively. Patients scheduled for a morning operating list receive no solids or milky drinks after midnight and patients scheduled for an afternoon list, or expected to travel to the operating theatre after 1200 h on an all-day list, are allowed to have a normal breakfast at 0800 h rather than be woken at 0600 h. These rules do not apply to emergency and trauma list patients where gastric

emptying may have stopped or the timing of surgery be uncertain.

We met initial resistance from some senior nursing staff, who were concerned that a degree of flexibility may be lost in re-organising lists on the day of operation, but our patients welcomed the new arrangement. Since these changes were introduced we have performed over 25 000 operations. We have not experienced any problems related to the new fasting policies, except that of convincing rotating junior staff not to cancel patients who have had a drink!

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J.K. MOORE

Pipeline contamination and oxygen quality monitoring

Since our own experience of oxygen pipeline contamination highlighted this potentially lethal problem, we were interested in the report by Dr Weightman and colleagues (*Anaesthesia* 1992; **47**: 500-2.). We would fully support their recommendations for 'surveillance' oxygen monitoring at the terminal of each pipeline branch, and for the disconnection of not-in-use pipeline supplied equipment. However, discussion with our engineers

suggests that maintaining air pipeline pressure below oxygen pipeline pressure (to act as a fail-safe in the event of cross connection) may not always be possible or practical.

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P.G. LAWLER
P.T.N. NEWNAM

Propofol and abreaction

I was pleased to read the first description of elective therapeutic abreaction using propofol (*Anaesthesia* 1992; **47**: 541-2). This is of particular interest to me as I used the very same technique in 1989 with some degree of success. The following is a description of what happened.

As the on-call anaesthetist I was asked to be present during an abreaction performed on a 21-year-old student nurse who had attempted suicide 2 weeks earlier; she had been an inpatient since. A previous attempt at abreaction had been made using diazepam 3 days previously, with no

anaesthetist present. This had been abandoned for two reasons; firstly the abreaction had been unproductive and secondly it was thought to be unsafe to continue considering the large dose of diazepam administered (45 mg intravenously over 1 h). Having seen abreactive type phenomena both on induction and recovery with propofol anaesthesia I suggested, after consultation with senior colleagues, that small incremental subanaesthetic doses of propofol may be a useful alternative to diazepam. We planned the case for 0900 h so the starvation period did not seem too artificial. The abreaction took place in the regular ECT room, hence an anaesthetic machine and full complement of anaesthetic drugs and equipment were present. The patient had consented the day before. Monitoring consisted of a pulse oximeter and automatic noninvasive blood pressure measuring at 5 min intervals. As in the case described, the lights were dimmed.

I gave small incremental doses of propofol up to a dose of 300 mg over a period of about 1 h, the dose being titrated against the patients response. On one occasion she appeared to fall asleep for a period no longer than 20 s but

her eyelash reflex was still present and no loss of airway occurred. The patient breathed air and at no point did her oxygen saturation fall below 95%. Her blood pressure stayed within normal limits throughout the procedure. The abreaction was felt to be useful because the patient had previously been resistant to psychotherapeutic techniques. She had several prolonged conversations relating to traumatic experiences involving family and friends, some going back to early childhood. After about 1 h she became quiet and indicated that she no longer wanted to continue. The patient returned to her ward and was discharged one week later.

Everyone involved felt that the procedure had been successful and that the fine line between creating a relaxed and ambient atmosphere conducive to psychotherapy and maintaining safety at all times was achieved.

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M.T. ALI

Blood pressure measurements and intravenous infusions

It is sometimes necessary to site an intravenous infusion in the same arm as that required for automated noninvasive blood pressure monitoring. This situation is unsatisfactory for several reasons. Whenever the blood pressure cuff inflates there is a rise in the distal venous pressure which causes blood to flow retrogradely up the infusion set tubing. This results in the infusion chamber filling up intermittently and also increases the chance of the tubing or cannula becoming blocked with blood clot. There may also be some uncertainty about the sequence and reliability of administration of drugs injected during, or immediately before a pressure determination. Finally, if the infusion is not turned off in anticipation of each measurement, there may be an increased risk of extravasation at the venepuncture site.

A simple method to avoid, or at least reduce, these problems is to route the proximal infusion set tubing *through* the blood pressure cuff so that it is sandwiched between the velcro layers. When the cuff inflates, the tubing is squashed flat and occluded just as the venous pressure in the arm begins to rise. Retrograde flow is thus automatically prevented at the appropriate moment. This easy manoeuvre is very effective if an intravenous administration set with fairly soft tubing is used. It solves an inconvenient and frustrating problem.

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C.M. WAIT

An unusual case of postoperative hoarseness

A 65-year-old male patient presented for abdominal surgery. After induction of anaesthesia with propofol and fentanyl, neuromuscular blockade was achieved with vecuronium and anaesthesia was maintained with 66% nitrous oxide in oxygen supplemented with 1.5% isoflurane. A 14 French gauge naso-gastric tube was passed with some difficulty after which the patient's trachea was intubated easily with a 9.0 mm tube. A triple lumen catheter was passed into the right internal jugular vein. The patient's lungs were ventilated with a Manley MP3 ventilator and anaesthesia proceeded uneventfully. In view of the difficulty experienced in passing the nasogastric tube, the surgeon confirmed its correct placement once the abdomen was open. At the end of the operation, reversal of neuromuscular blockade and extubation of the trachea proceeded uneventfully.

After the patient regained consciousness in the recovery room, he soon complained of marked hoarseness. There was no stridor and air entry was good to all zones of the lungs.

The SpO₂ remained at 98% with oxygen supplementation via a Hudson Mask. A chest radiograph had been ordered to check the position of the triple lumen catheter (Fig. 1).



Fig. 1. Chest radiograph showing loop of nasogastric tube passing into the right main bronchus and tip of nasogastric tube within the stomach.

This showed that the nasogastric tube had been passed down the trachea to the right main bronchus, had doubled back on itself to re-emerge through the larynx and thence passed to the stomach where its tip was clearly demonstrated. The nasogastric tube was hence withdrawn some 15 cm which was immediately followed by the restoration to normality of the patient's voice.

Accidental intubation of the trachea with a nasogastric tube is well recognised. This case demonstrates that even direct manual confirmation of the presence of such a tube in the stomach does not always exclude tracheal intubation.

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J.B. MITCHELL

Anaphylactoid reaction to topical chlorhexidine during anaesthesia

I wish to report an anaphylactoid reaction to topical chlorhexidine in a 12-year-old boy having revision hip surgery.

Previously anaesthesia had been uneventful but the anaesthetic drugs and surgical skin preparation used were not known. He was premedicated with diazepam. Anaesthesia was induced with fentanyl and propofol with 1% lignocaine and maintained with isoflurane, nitrous oxide, oxygen, vecuronium and increments of fentanyl. The surgical site was cleaned with chlorhexidine gluconate 0.5% in alcohol. Physiological variables were stable for 1 h, but then the airway pressure rose to 40 cmH₂O, oxygen saturation (SpO₂) decreased to 93%, and bilateral wheezing was heard. Blood pressure and heart rate were unchanged. Initially, we thought the tracheal tube had been displaced and the bronchospasm was caused by stimulation of the carina. The tracheal tube was repositioned, the inspired concentration of isoflurane increased and the lungs were manually ventilated. Shortly afterwards the blood pressure decreased to 70/25 mmHg, the heart rate rose to 140 min⁻¹ and marked skin erythema and facial oedema were noted. An anaphylactoid reaction was diagnosed. The isoflurane was discontinued, and he was given human albumin 30 ml.kg⁻¹ and clemastine (an H₁ antagonist) 0.5 mg intravenously. The blood pressure rose to 110/60 mmHg but the erythema, bronchospasm and oedema persisted and the SpO₂ remained at 92%. He was then given 3 µg.kg⁻¹ of adrenaline intravenously over 5 min. The bronchospasm and erythema resolved but 10 min later he again became hypotensive, erythematous and the oedema worsened. He was given more colloid, intravenous adrenaline and steroids. This pattern, in which the reaction would resolve with intravenous adrenaline but recur 10–15 min later, persisted. It was impossible to abandon surgery and over the course of 2 h he was given a total of 10 µg.kg⁻¹ of intravenous adrenaline. The initial reaction was not associated with anaesthetic drugs or blood transfusion, but none of the drugs used at induction were given again. The persistence of the reaction suggested a continuing exposure to the responsible stimulus and we thought that a topical

agent might be responsible. The skin that had been treated with chlorhexidine was cleaned with water, after which one more bolus of adrenaline was needed. After surgery the patient was admitted to the Intensive Care Unit and his trachea was extubated uneventfully the next day.

Postoperative skin scratch testing showed a weakly positive response to chlorhexidine 0.5% in alcohol but a positive urticarial reaction to a 1% solution. The results were equivocal for vecuronium and negative for all other drugs tested. The patient has had uneventful subsequent general anaesthesia including vecuronium.

Skin testing cannot categorically identify the cause of an anaphylactoid reaction but the results of these investigations and the pattern of the reaction suggest that topical chlorhexidine was responsible. There have been few reported cases of systemic reactions to topical chlorhexidine [1–3] and only one during anaesthesia [4]. This case highlights the importance of considering non-anaesthetic causes for anaphylactoid reactions during surgery.

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The 'hidden danger'

I recently ran a quiz at the Southern Society of Anaesthetists meeting at which advertisements of early copies of this and other anaesthetic journals were displayed, with essential bits of information blanked out. It was obvious that younger anaesthetists are unaware that the 'hidden danger' in operating theatres of the 1950s was static charge. With the introduction of antistatic precautions in the early 1960s, the danger of operating theatre explosions decreased despite the use of ether and cyclopropane still being widespread. These agents are now disappearing and some operating theatres are declaring themselves 'flammable free zones' with potential abandonment of antistatic precautions at a cost saving.

Can we really be so complacent that the danger of explosions in theatre has really passed?

An article by Gally in 1954 [1] pointed out the dangers posed by flammable gases produced by the gastrointestinal tract. In the paper he cites a case report from 1952 where anaesthesia was being induced for a mastoid operation by administration of thiopentone and gallamine followed by manual inflation of the lungs with 100% oxygen i.e. not so far removed from a modern induction. On lifting the rubber mask from the face, a fatal explosion took place and a postmortem showed extensive bruising of the whole of the gastrointestinal tract proximal to the stomach with concomitant rupture of the stomach. It was proposed that

gastric stasis had led to fermentation and manual inflation had forced oxygen into the stomach and the explosive mixture had tracked up the oesophagus into the mask. A static spark occurred as the mask was removed from the face thus igniting the mixture.

As long ago as 1980 gas analysis from the stomach of a patient with pyloric stenosis had revealed the following: carbon dioxide 56.0%; hydrogen 28.0%; methane 6.8%; air 9.2%; total combustible gases 34.8%.

Although Sellick's manoeuvre has been introduced since the 1954 article appeared, patients may swallow repeatedly during pre-oxygenation and allow mixing of oxygen with fermented gases. We thus have a couple of minutes during which the same conditions occur as they did in 1952 with the exception of antistatic precautions. If we remove this

last factor then what is to stop history repeating itself? How would we stand legally in this event bearing in mind that the danger has been known for so long?

Dr Gally's article, despite reporting this tragic event, is full of wit and humour and I recommend it to everyone.

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Reference

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Hysteria: a cause for opisthotonus

Professor Adams' interesting report of hysteria causing a failure to recover after anaesthesia (*Anaesthesia* 1991; 46: 932-4), prompts me to report a similar case of hysterical conversion following an elective repeat oesophageal dilatation.

The patient was a 49-year-old nursery nurse with a known history of a pharyngeal web. She had been depressed and was taking amitriptyline 150 mg at night, but was otherwise fit and well. She denied any previous anaesthetic problems. Routine pre-operative investigations were normal. The patient's old notes were unavailable; nevertheless the surgical team were confident of the diagnosis and so the operation proceeded. Premedication consisted of oral temazepam 20 mg, and anaesthesia was induced with propofol 120 mg, followed by atracurium 25 mg. The trachea was intubated and anaesthesia maintained with isoflurane 1% in nitrous oxide 67% and oxygen 33%. Following the procedure, when the patient was awake and breathing adequately, she was transferred to the recovery ward. Shortly afterwards she appeared to have a generalised tonic-clonic seizure, followed by a 10 min hypertonic stage during which she developed profound opisthotonus. This was relieved by an incremental dose of midazolam 7.5 mg at which point she opened her eyes and responded to simple comments. Her blood sugar and biochemistry were normal.

The patient subsequently suffered over 30 similar episodes lasting between 5 and 70 min. During attacks there was no change in the respiratory pattern, SpO_2 (97-98%) or blood pressure. The majority of attacks resolved spontaneously but prolonged events were treated successfully with bolus doses of midazolam (2.5-7.5 mg). Intravenous procyclidine 10 mg was used on two occasions but produced no discernible effect. Between attacks the patient was sleepy, but a full neurological examination was otherwise normal, with no signs of persisting hypertonicity. She was reviewed by a consultant physician who diagnosed

an acute dystonic reaction, probably drug induced. Centrally acting antidopaminergic drugs are usually implicated in such reactions, but no drug of this type had been administered. Propofol, however, has been reported to cause similar though short lived idiosyncratic reactions [1].

After 24 h the frequency and severity of the opisthotonus was increasing, associated with a rise in CPK to 980 IU, so the patient was transferred to the ITU. Only then were her old case notes finally located. These revealed that in addition to the history volunteered, she had attended the pain clinic on many occasions because of chronic backache. She had eventually been discharged because of the persistent psychological component to her symptoms. Moreover, her old anaesthetic charts revealed that she had experienced two previous episodes of seizures and opisthotonus lasting a few hours following general anaesthesia. On one occasion these were witnessed by a consultant neurologist who felt that they were hysterical in nature and would resolve spontaneously. We told the patient that we now had this information, following which she had no further episodes. She subsequently made a complete recovery and will be reviewed by the psychiatrists.

This patient showed the typical features of hysterical conversion described by Adams and Goroszeniuk. Unfortunately she also highlights the potential pitfalls of proceeding with surgery when old notes are unavailable.

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Reference

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Hypocapnia and mental function

I read with interest the original article by Jhaveri (*Anaesthesia* 1989; 44: 635-40) which concerned the effects of hypocapnic ventilation on mental function in elderly patients undergoing cataract surgery. The study concluded that these patients, when their lungs were ventilated to a mean PaCO_2 of 2.9 kPa for a mean period of 47.4 min, showed no impairment of psychological function as measured 4 days and 4 weeks after the operation.

The study gives no account of whether there was any

such impairment in the recovery ward or on the first postoperative day. It appears that further investigation is required to show that hypocapnic ventilation does not cause postoperative confusion in this important period. Until then, I would question the value of hyperventilating patients to such a low PaCO_2 , requiring fresh gas flows of up to 20 l.min^{-1} , when it has been shown that normocapnic IPPV anaesthesia lowers intra-ocular pressure and provides satisfactory operating conditions [1-3].

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Throat packs and airway protection

I was interested to read the letter from Dr Larard (*Anaesthesia* 1992; **47**: 638) on the use of throat packs in nasal and orosurgical cases.

I used to pack the throat routinely in these cases as I had been taught to do. I noted a virtually 100% occurrence of sore throats postoperatively and am aware of several cases in which failure to remove blood soaked packs at the end of surgery has compromised the airway. With the advent of plastic tracheal tubes with reliable cuffs, I therefore long ago abandoned the use of throat packs and now seldom get complaints of sore throat.

Like Dr Larard, I have now anaesthetised a very large number of such cases this way and have had no cause to regret my decision. The one practical point to note is that suction of the oropharynx at the end of surgery should precede the return of the swallowing reflex to prevent ingestion of blood. This is not, of course, life threatening, but it does tend to make patients sick.

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W. RYDER

Two Boyle's machines for inhalational induction of anaesthesia

A 72-year-old man was referred with a hoarse voice, stridor and dysphagia, worsening over several weeks, associated with anorexia and weight loss. He was a smoker with mild chronic obstructive airways disease, but was otherwise fit for his age. Pan-endoscopy was performed under general anaesthesia, and he was found to have a large supraglottic tumour with some subglottic extension. This anaesthetic was uneventful, a 5.0 mm cuffed nasotracheal (microlaryngoscopy) tube being passed without difficulty after inhalational induction of anaesthesia with halothane. Histology of the biopsy samples showed no malignancy, so microlaryngoscopy under general anaesthesia was repeated on two further occasions, the final sample revealing poorly differentiated squamous cell carcinoma. Unfortunately, after the third microlaryngoscopy, sudden drastic swelling of the region of the tumour worsened his respiratory obstruction until a decision was made to insert a Mini-Trach as an emergency auxiliary airway, pending a formal tracheostomy. In view of the histology it was felt by the surgeons that definitive laryngectomy should precede tracheostomy to prevent tumour spread, thus precluding the option of initial tracheostomy under local anaesthesia.

The problem that was thus presented to the on-call anaesthetists was to conduct anaesthesia safely in a man with two extremely narrow airways. One solution would have been to undertake an awake fiberoptic intubation under local anaesthesia. However, due to the distressed and anxious state of the patient and the risk of major haemorrhage into the airway, inhalational induction of anaesthesia followed by fiberoptic intubation was felt to be most appropriate.

After institution of full noninvasive monitoring and

establishment of large-bore intravenous access, inhalation induction was attempted using isoflurane in oxygen supplied through a Mapleson A system and face mask. The severe restriction to gas flow through the larynx, coupled with the mixing of inhaled anaesthetic gas with inspired air from the Mini-Trach, prevented the attainment of alveolar levels of isoflurane sufficient to induce deep anaesthesia, despite a concentration of isoflurane delivered from the mask of 5%. A second anaesthetic machine was then pressed into service to supply the same gas mixture to the Mini-Trach. This strategy allowed the attainment of a depth of anaesthesia sufficient to permit fiberoptic intubation during spontaneous ventilation. This whole process took nearly one hour, during which the surgeons were ready to perform an emergency tracheostomy if necessary.

Maintenance of anaesthesia during the process of laryngoscopy and intubation was achieved through the Mini-Trach and a Mathias oro-pharyngeal airway sealed into the mouth with *Sleek* tape. A 7.0 mm cuffed nasotracheal tube was then passed over the fiberoptic scope into the trachea and anaesthesia continued with nondepolarising neuromuscular blockade and positive pressure ventilation. Laryngectomy and formal tracheostomy were then performed.

To our knowledge, this is the first reported use of two Boyle's machines for the inhalation induction of anaesthesia.

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A. BANERJEE
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Patient misunderstanding

Dr Weightman's letter (*Anaesthesia* 1992; **47**: 632) reminds me of an incident which occurred when I was in regular anaesthetic practice. As I was leaving a ward, having performed a pre-operative assessment on a female patient I was to anaesthetise the following day, I heard the patient in the neighbouring bed ask 'What was the name of that doctor?' to which my patient replied 'Oh, he's not a doctor, he's just the anaesthetist'.

Since the patients whom Dr Weightman surveyed appear to have recognised that all anaesthetists are doctors, clearly we anaesthetists are greatly improving our communication!

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J. HICKEY

Book reviews

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MCQ companion to the textbook of anaesthesia

D. FELL, D.R. DERBYSHIRE, A.R. AITKENHEAD AND G. SMITH. Pp. ix + 276. Churchill Livingstone, 1991. £12.50.

Multiple Choice Questions (MCQ) are always in demand by doctors preparing for the Fellowship examinations and have been used by others as a method of keeping up to date. This volume contains 520 questions at a Part 1 FRCAnaes level, divided into topic sections: physiology, pharmacology, medicine and anaesthesia, and an examination section with two papers with questions in random order. The introduction contains helpful suggestions on answering MCQs and self-examination is facilitated by arranging the questions on one side and the answers with a brief explanation on the other.

The book's other role is, as the title suggests, to be a companion to the *Textbook of Anaesthesia*, second edition. Unfortunately, there were no offers of two for the price of one, so despite having the first edition readily available, the second edition had to be obtained, because references to the second edition could be traced in the answer section. References indicated a chapter rather than a page number so time was spent reading additional information. Two other books are used in the answer section; these are *Davidson's Principles and Practice of Medicine*, 15th edition, 1987, and the *British National Formulary*, 20 September 1990. There are a few questions in each section which are unreferenced, and these are not all 'false' answers.

Writing MCQs is not an easy task, but some questions pose a double negative and the stem and the item do not form a statement. Pharmacology changes rapidly and there are no complete questions (stem and items) on propofol or isoflurane, but two each on methohexitone and dantrolene. Abbreviations are used in the text without being explained in full (there is no index or appendix) and SI units are not used for cylinder pressures.

The topic sections each contain 100 questions. Physiology includes a little anatomy, but mainly the respiratory, cardiovascular, nervous, gastrointestinal, renal and endocrine systems, and blood, fluid and electrolytes. Pharmacology has a balanced number of questions related to anaesthetic drugs (local anaesthetics, neuromuscular blocking drugs, analgesics, intravenous and inhalational anaesthetics), there are questions on basic principles and adverse reactions and almost a fifth relating to cardiovascular drugs. Questions in medicine follow a similar pattern to physiology and include resuscitation. Anaesthesia is a varied section and covers a wide range of subjects including surgery. The two test papers focus on medicine, anaesthesia and pharmacology with a few questions on physiology, surgery and anatomy.

This collection of MCQs will be helpful to candidates preparing for the Part 1 Fellowship in Anaesthesia (DA), but, as the authors indicate, a bank of questions requires constant revision to reflect current practice. It is not the content of the questions *per se*, but the experience of learning how to utilise information to answer questions correctly which is relevant. This could be achieved by working through the MCQs in conjunction with the three reference books. (Will any publisher make an offer of a multiple purchase bargain price?).

A. HOLDCROFT

The ageing surgical patient: anaesthetic, operative and medical management

Edited by D.L. CROSBY, G.A.D. REES AND D.G. SEYMOUR. Pp. 457. John Wiley & Sons, 1992. £65.

This multi-author book, which is aimed at a wide group of readers, namely surgeons, anaesthetists, hospital doctors, nurses and paramedics, collates information on the anaesthetic and surgical management of surgical conditions in the elderly.

The introductory chapter gives a precis of the general principles of such management, supported, as are all the chapters, by an extensive reference list. Demographic statistics are included in addition to a great deal of physiology specific to ageing. The rationale behind surgical treatment in the elderly is discussed, together with an assessment of the risk factors involved. This is augmented later in the book by a personal view of the risks of anaesthesia and surgery by J.N. Lunn.

The majority of chapters are written by both an anaesthetist and a surgeon and have a very practical emphasis. There are particularly well written chapters on orthopaedics and rehabilitation, vascular anaesthesia and surgery, cardiac and neurosurgical management together with the surgical treatment of acute pain. The section on Intensive Care is especially relevant, supporting the more optimistic approach to care in the elderly, and includes assessment, length of stay and outcome analysis. Medicolegal and ethical aspects are discussed separately and there is useful information on confidentiality and obtaining consent to treatment applicable to all age groups. The final chapter raises topics for possible further research, discussing screening, audit and the role of computers with respect to elderly patients.

The photographs, diagrams and X ray reproductions are more relevant to surgical practice than anaesthesia, although anaesthetists should find the tables in chapter one on age-related complications and risks interesting.

The Ageing Surgical Patient should not principally be viewed as a reference text (despite its comprehensive index enabling selection of specific topics) or a tome to be skimmed through. This is a work to be read from cover to cover as each chapter contains information on general management in addition to its specialist bias, e.g. fluid balance, temperature control and pre-operative assessment and preparation.

With its multidisciplinary and holistic approach, the Editors have succeeded in producing a text which is of general interest to anaesthetists in the overall management of elderly surgical patients.

C.J. KNICKENBERG

Standards of care in anaesthesia

T.H. TAYLOR AND D.R. GOLDHILL. Pp. x+186. Butterworth, Heinemann, 1992. £29.50.

I enjoyed reading this book, and so will you. But before you start on the 173 pages of anaesthetic text, read the foreword by Stanley Feldman, and Chapter 9, by Simon Taylor, Barrister at Law. The foreword explains why the book needed to be written. 'Mandatory regimes strip doctors of the freedom to make decisions based on medical knowledge, anaesthetic training and technical skills'. It is much better to define the objectives and limits of proper safe practice which will produce a minimum acceptable standard of care, and leave the method of achieving them to the clinician. Thank goodness for a book which avoids dogma, but achieves exactly what its authors set out to do—provides the information on which sensible decisions can be made.

Mr Taylor, in 'Standards of care: the good, the bad and the acceptable' examines the limitations which should be put upon the use of the book in a medicolegal context. He points out that there are differences between what is best and what is acceptable, but that only on a case-by-case basis can it be determined when the best (which we aim for) or the acceptable (which I hope we achieve) sinks to being negligent. In other words, it is very important to realise that *Standards of care in anaesthesia* is not a list of rules and regulations.

The eight chapters which make up the body of the book follow an anaesthetic through in a logical manner. Two essential chapters on pre-operative assessment are followed by one on pre-operative considerations. These are followed by 'Care in the operating theatre' and 'Monitoring and recording'. This is all good common sense material, well written and well argued. How sensible to find a brief discussion on surgical and anaesthetic risk, preceding pre-operative assessment where both the time and place of the assessment are considered under various circumstances. And how honest to say that the patient's notes from previous procedures should be examined 'if they are available'. By implication, as we all know, they frequently are not. This solid clinical material leads on to what is a virtual guideline about how to run an anaesthetic room, and then a review of the various national standards of minimal monitoring.

Chapter 6 covers care of the staff and the working environment. This considers pollution, sharps, cross infections, fatigue and stress, noise and drug abuse. The authors obviously like quiet operating theatres, with muted conversations, telephones and pagers. Many would disagree with some of these, since at night a mobile phone in the operating theatre may be the only way to keep in contact with the emergency team, and if it wasn't for a bit of music, a lot of plastic surgery would drive me mad. The book finishes with a relatively short account of care in the recovery

period, which should be shown to the hospital accountants, and a chapter on standards and audit.

This is an excellent book. I shall not put it on the departmental shelf because it might get pinched. I want it where I can find it, as a source of good practice, common sense, and reassurance when unrealistic and impracticable ideas of 'how it must be done' are floated by colleagues, or colleges.

R. WELLER

Researches practical and physiological on etherization

N.I. PIROGOFF AND B.R. FINK. Pp. 76. Wood Library Museum, 1992. \$12.00

Nikolai Ivanovitch Pirogoff (1810–1881) is remembered by surgeons as an innovative anatomist, operator, and teacher; but in the histories of anaesthesia he is recorded only as the pioneer of the rectal administration of ether. On the evidence of this small book alone, this is a great injustice.

Pirogoff studied medicine at Dorpat (now Tartu), graduating in 1832. He was professor of surgery there from 1835 to 1841, and then proceeded to the chair at St Petersburg. He started experimenting with ether early in 1847, and first administered it to a patient by inhalation on 14 February. This book was published at the end of May. It is divided into four parts. The first is a wide-ranging discussion during which Pirogoff describes his reaction to the introduction of general anaesthesia. Elaborating the two 'one-liners' that are part of our folklore, this presents a thoughtful and sensitive attempt to grapple with a completely new, alarming, but potentially epoch-making phenomenon. On analogy with alcoholic intoxication, Pirogoff was concerned that ether might have subsequent excitatory effects. Also for him, 'a surgeon steeled by courage, judgement, and experience to disagreeable and unwelcome screams and reactions to pain of his patients, an operation performed on a person robbed of feeling and consciousness is bound to be repugnant.' Usually, insensibility supervening during a major operation in which the patient was, 'a moment earlier happy, jolly, and full of life,' presaged death from 'nervous apoplexy' or the entry of air into the veins; but with an already unconscious patient, how could one know when the danger point had been reached? How could an unconscious patient cooperate, if required to change the posture of the operated limb, to hold his breath, or to breathe deeply? Such observations help answer the question, 'Why did it take so long?' And Pirogoff is aware of this, for he continues, 'who could accuse us of having hesitated overlong, given that we regarded our patients' cries of pain as inseparable from an operation and as signs of an appropriate reaction?' But having overcome his scruples and gained some experience, he perceptively sounds a chord that reverberates loudly today, observing that ether could, in one respect, utterly transform the entire practice of surgery, by removing the hesitation to perform palliative operations. Surgeons had hitherto been reluctant to inflict pain if the sole end had been temporarily to prolong life or mitigate agony. But he had performed several palliative operations which he would not previously have contemplated, with immense relief; for example, the amputation of an osteomyelitic femur which was producing such a profuse stinking suppuration that it soaked the mattress in a few hours, and disseminated a stench unbearable to the patient and the attendants alike.

Pirogoff describes the clinical progress during ether inhalation. He was concerned that his patients often hallucinated, and he soon became aware of the inadequacy of the

Charrière inhaler, and designed his own, but he continued to regard the effects of inhaled ether as unpredictable, and advocated a preliminary test inhalation. He concluded this section with a description of his own inhaler, which he also illustrates, and he draws attention to some important practical points in its use.

The second section describes Pirogoff's experimental attempts to determine the mode of action of ether. He applied it directly to the nerves of animals, to their spinal cords, and he introduced it into the blood stream, directing it centrally and peripherally, and into the trachea and the rectum. He concluded that the conditions necessary for sleep and insensibility were that the ether vapour had to enter the blood, and make contact with the nervous system, the central parts of which appeared to be more sensitive than the peripheral. He also observed that liquid ether was not equivalent to ether vapour, since it produced sudden death. He described four degrees of etherization, but almost entirely in pharmacological terms. He speculated that ether acted either physically, the vapour compressing the nervous tissues, or chemically, transforming the mutual relations of the atoms in the nerve fibres.

In the last section Pirogoff describes his method of rectal etherization. He allows priority to Magendie, although his own may have been an independent innovation. After an unsuccessful experiment with liquid ether, he had devised a vaporizing syringe with a hot water jacket, which he describes and illustrates. Usually the vapour derived from two ounces of ether was sufficient to produce narcotization within 3 min, and without excitement. The effect lasted longer than after inhalation, and the results were so much better that he was convinced that the rectal route would entirely replace the inhalational for most cases. In a later communication he reported on the use of rectal etherization for operations involving the face and air passages.

To compare Pirogoff's approach with John Snow's is irresistible. Strongly influenced by Magendie, Pirogoff's pharmacology was precisely of his own time, anatomical pharmacology, the search for sites of action. It became the pharmacology of the great school of Dorpat, of Buchheim, Schmiedeberg, and the other luminaries of the nineteenth century. But Pirogoff shows no evidence of understanding the application of the basic sciences to the design of an effective vaporizer. John Snow, on the other hand, starting from the same base, but influenced initially by Dalton, concerned himself with such twentieth century topics as saturated vapour pressures, anaesthetic uptake and the correlation of blood levels with degrees of narcotization, the relationship between potency and blood solubility, and the effects on cellular respiration.

Gratitude is due to Dr Fink for his most readable translation of this rare book, and to the publishers for making it available. He has provided a short analytical introduction, and reprinted Professor Ole Secher's biographical essay on Pirogoff from *Anaesthesia* 1986. Everyone interested in the history of anaesthesia should not only read, but study, this brief but important book.

D. ZUCK

Atlas of procedures in anaesthesia and critical care

J.F. BUTTERWORTH. Pp. 239. W.B. Saunders, 1992. £18.50.

This book covers, in illustrated form, the practical procedures which are commonly performed during anaesthesia and in the intensive care unit. There are sections on airway control, vascular cannulation, regional anaesthesia and positioning the patient on the operating table. It is comprehensive and even the 'simple' procedures, such as main-

taining an airway and intravenous cannulation, are described. The only important omission in a book covering intensive care procedures is the technique of chest drain placement.

The single author describes his own way of doing things, which will not reflect every readers' methods. However, this type of text should not give too many alternatives which only serve to confuse the beginner; more comprehensive sources are referenced where relevant. The practical approach is exemplified by the description of distal nerve blocks in the arm to supplement incomplete or slowly evolving brachial plexus blockade. There are a few errors, such as an incorrectly labelled subclavian artery in a diagram of supraclavicular nerve block; some transatlantic differences such as a description of a cannula-through-needle technique for central venous cannulation; and the illustrations of the limbs are not confined to one side, which is potentially confusing. A diagram of the grading of direct laryngoscopic views would be helpful, and beginners should probably be advised to gown as well as glove for Seldinger techniques of central venous cannulation, as well as spinal and epidural anaesthetics.

This book would be useful in departmental libraries for reference by the new, as well as the not-so-new anaesthetist, and also for those, such as ODAs and intensive care nurses, who are expected to assist anaesthetists with these procedures. At under £20, it also represents excellent value for money.

M. KINSELLA

Adult respiratory distress syndrome

A. ARTIGAS, F. LEMAIRE, P.M. SUTER AND W. ZAPOL. Pp. 527. Churchill Livingstone, 1992. £90.

This multi-author textbook, written by well known experts in their fields, consists of nine sections with 47 chapters in 527 pages. It is an interesting and detailed review. The first section highlights the clinical aspect of the disease, and the first chapter provides a useful table setting out a bedside classification of ARDS which is then used elsewhere in the book. The next two chapters are excellent accounts of ARDS in haematological disorders and pulmonary infection in ARDS.

Section two comprises four chapters devoted to morphological changes, with much erudite histological detail in the first two. Section three consists of eight chapters devoted to pathophysiology, four of which are particularly interesting. Two of these chapters, written by the same author, are compulsory reading—the first includes a lucid account of mediator cascades and the second concerns the arachidonic acid/cyclo-oxygenase sequence written in the same illuminating style. The third chapter is an excellent account of the role of the oxygen radical in ARDS and also gives details of some anti-oxidants. The fourth is concerned with the possible role of proteases and gives a graphic account of their release from neutrophils. Section 4 has four chapters devoted to lung water content and the circulation, and includes a useful chapter on the pathophysiology of pulmonary oedema. Section 5, on the pulmonary circulation and cardiac function, consists of seven chapters the first of which includes useful details of the physics of pressure measurement.

The following chapters are lucid accounts of hypoxic pulmonary vasoconstriction, written by the only British contributor; the pulmonary circulation in ARDS which includes a useful table summarising the possible mechanisms for the increased pulmonary vascular resistance, and pulmonary angiography. The chapters on the effect of

pulmonary hypertension on right ventricular function and the haemodynamic consequences of acute respiratory failure in the presence of coronary artery disease are compulsory reading.

Section 6 is devoted to pulmonary function with some useful chapters on hypoxaemia, ventilation—perfusion inequality, and the study of gas exchange as a prognostic factor in ARDS which provides some interesting yardsticks. The chapter on the lung pressure volume relationships during mechanical ventilation is well illustrated with a number of useful pressure volume loops.

Section 7 on mechanical ventilation contains a number of good reviews on various modes of ventilation and their consequences. The chapters on differential ventilation and high-frequency ventilation, should be read by all clinicians not familiar with these techniques. Chapter 41, however, written by the same author as the review, is packed with complicated experimental data which is totally unstimulating for the general reader and could well be removed or amalgamated in an abbreviated form in the review chapter.

Section 8 is concerned with therapy, and includes good chapters on extracorporeal support, vasodilators, eicosanoids and bronchodilators. Unfortunately, however, there is no mention of the more recent developments such as the role of nitric oxide.

Section 9 contains two chapters looking at prognosis and the future. The first outlines the difficulties involved in designing clinical trials and should be read by all those planning research projects. The final chapter reviews the diagnosis of ARDS and includes a useful table showing the diagnostic criteria used in various centres.

This textbook is essential background reading for all those interested in the subject and upon which to build a base for understanding the current new developments, even though there is overlap in certain areas with books published in the mid 1980s. The disappointment lies in the time lag between writing and publishing, which has meant there is a dearth of references to recent advances in the 1990s.

D.R.G. BROWNE

Errata

Anaesthesia, 1992, Volume 47, page 714

Hazards of supplementary oxygen

J. HARRISON, M. GHURYE AND G. B. SMITH

The word 'not' was omitted from the final sentence of the above letter. The sentence should read:

Finally, whilst we would not state that all patients who have upper gastro-intestinal anastomoses should be denied respiratory support in the form of mask CPAP, we would now, in the presence of an established leak, elect to intubate the patient's trachea and provide CPAP via a tracheal tube.

Anaesthesia, 1992, Volume 47, page 718

Evaluation of the E25 for the Level 1 blood warmer

J. M. B. SMALLMAN

The figure legend in the above letter should read:

Fig. 1. Relationship between flow and issuing temperature of the E25 (◆), D50 (◻), and Fenwell (◻) warmers.

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Edited by

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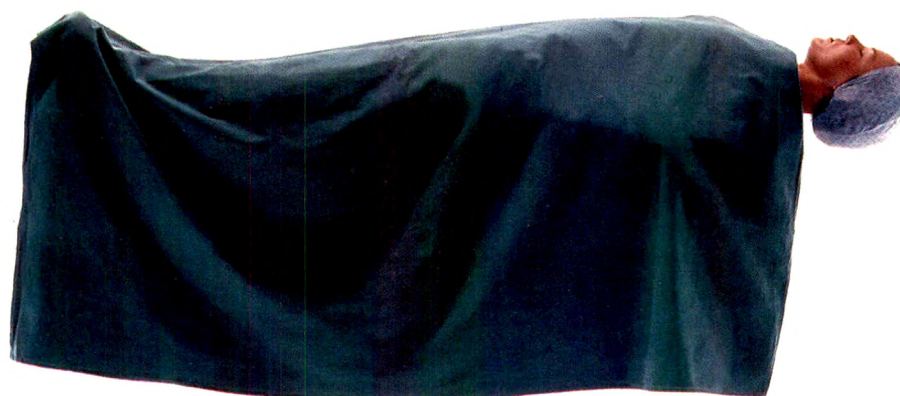


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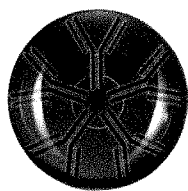
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PRESENTATION Centoxin is a solution for intravenous infusion after dilution with normal saline. Each vial contains:

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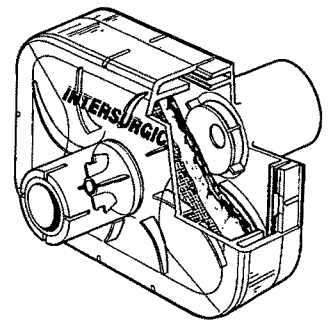
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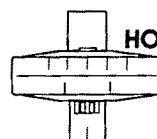
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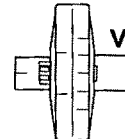
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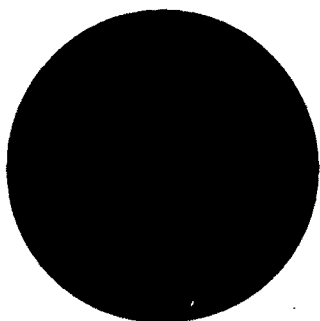
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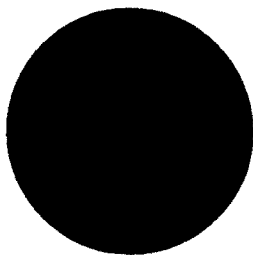
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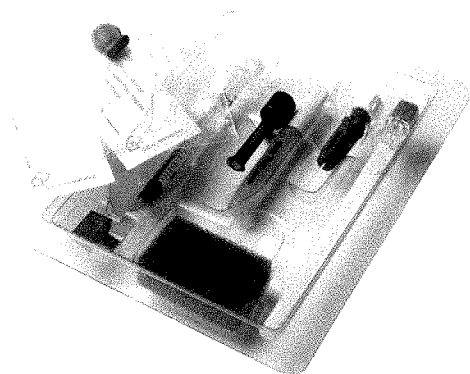
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Editorial

A safe haven or a dangerous place—should we keep the anaesthetic room?

Anaesthetic rooms have been a constant feature of British operating theatre design. However, there are many countries where they are not used, and their value in this country has recently been questioned [1, 2]. The first successful public demonstration of ether anaesthesia took place in October 1846 in the Massachusetts General Hospital in Boston, when Morton induced anaesthesia in the operating theatre in front of a crowd of spectators. The first mention of a separate room to anaesthetise a patient appears to be in the *Cornhill Magazine* in 1860, which, in reviewing UK practice states 'the patient is inhaling chloroform in an adjoining apartment, under the care of a gentleman who makes that his special duty ... the unconscious patient is brought in ... and laid upon the operating table' [3]. In America, similar practices were occurring in 1873 in the Massachusetts General Hospital. Osborn, in 1976 wrote that 'chloroform should be always administered ... in a small room adjoining the theatre previously to the patient being brought in for operation as he does not then become excited and is more quickly brought under the influence' [4]. St. Georges Hospital operating theatres, planned in 1894, were opened in 1896 where 'each theatre has an adjoining room to which the patient is first taken and placed under anaesthetic before being removed to the theatres. He is thus spared the sight of many things that might otherwise shock and distress him' [5]. This concept was universally accepted and operating theatres worldwide were designed to include an induction room.

The importance of a quiet room for induction was mentioned again in 1914 by Robert Ferguson [6], and Buettner in 1924 [7]. A variety of reports relating to the psychological preparation of surgical patients discuss the design and ambience of the ideal anaesthetic room: e.g. 'peaceful, warm and light, not gaudy' [8]. In 1937, a publication from the Ministry of Health recommended an anaesthetic room per operating theatre with a floor area of 13 m² and direct entry to theatres from this induction room [9]. In 1947, the anaesthesiologists of Illinois stated that there was a trend towards the use of a separate room for anaesthetising which could enhance patient turnover [10]. However, the US Public Health Service publication 8 years later did not include anaesthetic rooms in its recommendations [11], and in the 1970s their use declined and eventually disappeared from building programmes in the USA. In contrast, anaesthetic room design has featured in British literature throughout the 1950s and 1960s [12-15].

The original role for anaesthetic rooms as a pleasant, quiet, calm place, in which the patient is shielded from 'sights which may shock him' has been suggested to be no longer valid if patients are adequately prepared [2]. Additional benefits claimed for the use of anaesthetic rooms are increased patient turnover and the ability to have the time to supervise trainees. Local anaesthetic procedures can be performed in the anaesthetic room, without delaying the progress of the operating list, and

delays in the collection of patients from their wards and the preparation or cleaning of operating theatres can further be avoided. Parents can accompany their children into the anaesthetic room, while they would not be permitted into the operating theatre. Adequate working surfaces, and proximity of all drugs and equipment, as well as reliable, dedicated anaesthetic help, are all quoted as advantages by protagonists of the anaesthetic room.

Arguments against anaesthetic rooms are those of increased capital cost and risk to the patient. The building of an anaesthetic room and duplication of monitoring equipment, which, if not provided, will mean inadequate monitoring at the most crucial time of an anaesthetic, is expensive. The disconnection and possible loss of monitoring, infusions, and other equipment during transfer of the patient, especially if the latter is unstable, is considered dangerous. Further, the continuity of monitoring, which induction in the operating theatre allows, will facilitate the keeping of a continuous anaesthetic record. Patients can position themselves on the operating table, reducing the risks of moving an unconscious patient. More quantitative help is available in the operating theatre in case of problems.

Loder reported the results of a questionnaire on anaesthetic rooms completed by those attending the annual meeting of the Association of Anaesthetists of Great Britain and Ireland in Harrogate in 1963 [16]. Dr S. Kanagansundaram and ourselves repeated this exercise in 1991, when the venue was once again in Harrogate. Despite a low response rate (exactly 100 of approximately 400 questionnaires issued), there were some very clear views. Consultants formed 96% of responders and two thirds of these worked in nonteaching hospitals; 94% had anaesthetic rooms available in almost all operating theatres (exceptions included day surgery, casualty, minor operations, obstetric and dental theatres) and 96% used the anaesthetic rooms all or nearly all of the time on routine operating lists.

Reasons for finding the anaesthetic room desirable were, in order of priority: 86% considered it to be very important in patient privacy and comfort, 77% valued drugs and apparatus being nearby, 71% appreciated anaesthetic independence, and 68% found the anaesthetic room useful for regional anaesthesia. An anaesthetic room for improving turnover was important to 57%, while storing equipment and teaching is considered a priority by less than half. Only 7% of the anaesthetists responding to the survey never anaesthetised in the operating theatre. Of the remainder, 80% induced emergency Caesarean sections in the operating theatre, while 52% induced elective Caesarean sections there. Two-thirds induced anaesthesia in emergencies of ASA grade 3-5 in the operating theatre, while only one-third do so on nonemergency patients with the same ASA grading.

Reasons given by those anaesthetists inducing in the

operating theatre were ranked as follows: 73% induced there if they required continuous monitoring, 68% in cases of surgical emergencies, and 67% if difficulty in the transfer of the patient from the anaesthetic room to the operating theatre was anticipated. Inadequate monitoring was only an indication to 43% of anaesthetists to anaesthetise in the operating theatre. Other reasons given to anaesthetise in the operating theatre included intensive care patients already monitored or difficult to transfer, gross obesity, neonates, patients with respiratory obstruction, HIV-positive patients, or minor operations.

The level of apparatus in the anaesthetic room met the Associations' minimum monitoring standards in only 54% of responders, 4% were unsure, and 42% had inadequate monitoring. Monitoring equipment was regularly transported between the operating theatre and the anaesthetic room by 29% of anaesthetists, sometimes by 4% and never by 69%. To the question whether the anaesthetist would be prepared to give up the anaesthetic room if peace and quiet could be ensured, 68% said no and 28% said yes; 52% of anaesthetists would not give up the anaesthetic room under any circumstances, 27% were undecided and 20% said they would.

From this survey it can be seen that despite only about half of anaesthetists having minimal monitoring available in the anaesthetic room, 94% used the room for all or nearly all elective inductions of anaesthesia. The anaesthetic room was considered very desirable for peace and quiet by 86% of anaesthetists, a feature which has been shown to make little difference to the patient's level of anxiety [2]. Perhaps it is the anaesthetist who requires peace and quiet, since 71% considered 'anaesthetic independence' to be important and only 28% would give up the anaesthetic room if this could be ensured. A high percentage of anaesthetists did induce patients in the operating theatre for certain indications, but the overall consensus was that British anaesthetists want to keep their anaesthetic rooms.

It is likely that the hospital administrators and those who wish to further control budgets will continue to question the need for anaesthetic rooms in future operating theatre plans. Anaesthetists will need more 'hard data' to justify their continued insistence on their 'quiet havens' in this time of financial constraints. 'Just because I like them' may not be a good enough reason

in the future. Perhaps the rapidly increasing evidence of the advantages of regional anaesthesia in the reduction of morbidity and prevention of chronic pain will save the anaesthetic rooms for UK anaesthetists?

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The relationship of blood lactate concentrations, oxygen delivery and oxygen consumption in septic shock and the adult respiratory distress syndrome

G. R. NIMMO, S. J. MACKENZIE, S. W. WALKER, J. CATNACH, M. NICOL, I. R. ARMSTRONG AND I. S. GRANT

Summary

Tissue hypoxia is thought to be pivotal to the development of multiple organ failure, but cannot be measured directly in clinical practice. We assessed the relationship between initial arterial blood lactate concentrations and the presence of the phenomenon of delivery-dependent oxygen consumption, both of which may indicate tissue hypoxia. Twenty-three critically ill patients with septic shock and adult respiratory distress syndrome were studied prospectively and allocated to one of two groups according to blood lactate concentrations. In group 1, blood lactate concentration was less than the level widely accepted as significant (2 mmol.l^{-1}); in group 2, the concentration exceeded 2 mmol.l^{-1} . In both groups, resuscitation with colloid, blood and vasoactive drugs resulted in significant increases in oxygen delivery; in group 1 ($n = 13$), mean (SEM) oxygen delivery increased from $484 (36)$ to $730 (44) \text{ ml.min}^{-1}.\text{m}^{-2}$ ($p < 0.005$) and in group 2 ($n = 10$) from $550 (54)$ to $780 (54) \text{ ml.min}^{-1}.\text{m}^{-2}$ ($p < 0.05$). In neither group was there a significant change in oxygen consumption. However, there were individuals in both groups who exhibited pathological delivery dependence. This suggests that the absence of hyperlactataemia does not preclude delivery dependence of oxygen consumption with the attendant potential for tissue hypoxia.

Key words

Shock; septic.

Lungs; respiratory distress syndrome.

Metabolism; lactate.

Oxygen; consumption.

Elevated arterial blood lactate concentrations have long been recognised as being a consequence of tissue hypoxia [1, 2], and of having significant prognostic importance [3–5]. However, there are many causes of hyperlactataemia other than tissue hypoxia; these include hyperglycaemia, hypocapnia, toxins, liver failure and some malignancies [1, 6]. It is generally accepted that hyperlactataemia in patients with acute cardiorespiratory failure results from a combination of overproduction of lactate in hypoxic tissues and of reduced clearance and metabolism [6]. The liver and kidneys may become net producers of lactate in shock [7]. The cardiorespiratory findings in septic shock and adult respiratory distress syndrome (ARDS) may include increased cardiac output and oxygen delivery. Despite the presence of an apparently adequate oxygen delivery there is an impediment to normal tissue oxygen utilisation which has long been recognised

but is still not fully understood [8, 9]. It has been shown that, by increasing oxygen delivery in these patients using fluids, blood or vasoactive drugs, oxygen consumption ($\dot{V}\text{O}_2$) may increase even when oxygen delivery exceeds the $330 \text{ ml.min}^{-1}.\text{m}^{-2}$ below which such a relationship is thought to be physiological [10–13]. Delivery-dependent oxygen consumption at these levels is a pathological state which is in stark contrast to the norm. Recent work has suggested that elevated blood lactate concentrations characterise those patients with shock or ARDS who exhibit delivery-dependent $\dot{V}\text{O}_2$ [14, 15] although we have previously shown that hyperlactataemia is not a universal finding in shock [16]. We have therefore re-examined the association between blood lactate concentration and delivery dependence in the critically ill. During resuscitation of 23 critically ill patients with septic shock and ARDS, serial cardiorespiratory profiles with concurrent blood lactate

G.R. Nimmo,* MRCP, Research Fellow S.J. Mackenzie,* FRCAnaes, Registrar, S. Walker,† MA, DM, Senior Lecturer, J. Catnach,* MB, SHO, M. Nicol,† BSc, FIMLS Research MLSO, I.R. Armstrong,* FFARCS, Consultant, I.S. Grant,* FRCP, FFARCS Medical Director.

*Intensive Therapy Unit, Western General Hospital and †Department of Clinical Chemistry, Royal Infirmary, Edinburgh, Scotland, U.K.

Accepted 19 March 1992.

concentrations were obtained and the influence of these on the relationship between oxygen delivery and consumption studied.

Methods

We studied 23 patients who required intensive care, invasive haemodynamic monitoring and mechanical ventilation for the management of septic shock ($n = 21$) and ARDS ($n = 23$). The study was reviewed and approved by the local ethics committees. Septic shock was defined as mean arterial pressure (MAP) < 70 mmHg with oliguria, despite controlled volume expansion to an optimal pulmonary artery occlusion pressure (PAOP), in the presence of positive bacteriology. ARDS was defined as severe hypoxaemia which required mechanical ventilation with $F_{IO_2} > 0.5$ to maintain $P_{aO_2} > 10$ kPa; diffuse infiltrates on chest X ray; shunt ratio $> 30\%$; PAOP < 18 mmHg; and the presence of a known risk factor.

All patients were sedated with continuous infusions of papaveretum $3\text{--}5$ mg.h $^{-1}$ and midazolam $1\text{--}3$ mg.h $^{-1}$, and the muscles were paralysed with atracurium. No changes in rates of infusion of these drugs were made during the study period, and the only adjustments to ventilator settings were of F_{IO_2} . All patients were studied during the first 6 h of resuscitation following admission to the intensive care unit. Femoral arterial and thermodilution pulmonary artery flotation catheters (PAFC: Swan-Ganz, Baxter/Edwards, Irvine, CA) were used in all patients for measurement of mean arterial pressure (MAP), mean pulmonary artery pressure (PAM), end-expiratory pulmonary artery balloon occluded (wedge) pressure, and mean right atrial pressure (RAP), all zeroed to the phlebostatic axis. Cardiac output was determined by thermodilution in triplicate using 10 ml dextrose 5% at a temperature of $< 10^\circ\text{C}$. Continuous ST segment analysis was performed using, and heart rate measured from, 5 lead ECG monitoring (7200 series bedside monitors, Marquette Electronics, Milwaukee, WI). Samples of mixed venous and arterial blood were taken for blood gas analysis within 1 min of the last cardiac output measurement. Haemoglobin concentration and mixed venous and arterial oxyhaemoglobin saturations were measured using an oximeter (Ciba Corning). The oxygen content of arterial (CaO_2) and mixed venous (CvO_2) blood, and oxygen delivery and consumption, were calculated using standard formulae. By convention, cardiac output is indexed for body surface area, and we calculated oxygen delivery as the product of CaO_2 and cardiac index (CI). Oxygen consumption (also indexed to body surface area) was calculated using the inverse Fick relationship: $\dot{V}O_2 = \text{cardiac index} \times \text{arteriovenous oxygen content difference}$.

At the time of arterial sampling, blood was taken for lactate measurement. A volume of 1.5 ml of blood was placed into preweighed glass tubes containing 5 ml of cold (4°C) 5% perchloric acid, immediately refrigerated and subsequently analysed. Lactate concentration was measured using a Cobas Bio (Roche) centrifugal analyser, monitoring the rate of reduction of NAD to NADH $^+$ fluorimetrically at an excitation wavelength of 340 nm in 0.5 M glycine buffer (pH 9.6) in the presence of 0.2 M hydrazine hydrate and lactate dehydrogenase. Lactate standards from 50–1200 mmol.l $^{-1}$ were used. Whole blood lactate concentration was then calculated after correcting

for dilution of the blood sample with 5% perchloric acid. The assay had a sensitivity of 0.05 mmol.l $^{-1}$ and a coefficient of variation of 5.8–7.9% depending on the concentration range. We have previously validated the use of arterial rather than mixed venous samples for lactate measurement [17].

Following baseline measurements, further controlled volume expansion was performed in all patients after comparison of the haemodynamic profile with previously described therapeutic goals: MAP > 80 mmHg, CI > 4.5 l.min $^{-1}$.m $^{-2}$ and oxygen delivery 600 ml.min $^{-1}$.m $^{-2}$ [18, 19]. Colloid and blood were used, depending on haemoglobin concentration. After volume expansion, all patients received combinations of vasoactive drugs (Table 1), depending on the haemodynamic profile, in doses titrated against the observed haemodynamic effects with the intention of reaching the defined therapeutic goals. The baseline variables were compared to the variables associated with maximal oxygen delivery within the first 6 h of resuscitation. Patients were allocated into one of two groups on the basis of the initial blood lactate concentration (group 1, lactate < 2 mmol.l $^{-1}$; group 2, lactate > 2 mmol.l $^{-1}$). This threshold for significant hyperlactataemia is that described by several investigators [2, 20].

Statistical analysis

All results are expressed as mean (SEM). Baseline data from the two groups of patients were compared by means of the Wilcoxon rank sum test, and the paired data obtained before and after resuscitation were compared with the Wilcoxon signed rank test. Differences were considered significant at $p < 0.05$.

Results

The 23 patients (11 female) were each studied on one occasion. The mean age was 55 years (range 36–81). Clinical data, including admission APACHE II scores, are shown in Table 1, and haemodynamic and oxygen transport data are detailed in Tables 2 and 3. Baseline measurements were compared in the two groups. Heart rate, oxygen delivery and lactate concentrations were significantly higher in group 2.

Following resuscitation, there were significant comparable increases in cardiac index, mixed venous oxygen saturation and oxygen delivery, and decreased oxygen extraction, in both groups. There was a significant increase in MAP ($p < 0.05$) only in group 1. There were no significant changes in mean $\dot{V}O_2$ or lactate concentration in either group, although there were marked individual changes in $\dot{V}O_2$ in some patients in both groups (Figs 1–4). There was one death during the study period (patient 16). In group 1, six of 13 patients survived to leave hospital; three out of 10 survived in group 2.

Discussion

It has been recognised for many years that inefficient utilisation of oxygen is a fundamental defect in septic shock [8]. This finding has been confirmed recently, and a similar situation has been described in ARDS [12, 19]. The pathophysiological abnormality is characterised by depen-

Table 1. Clinical details: group 1.

Patient	Age	Sex	Primary diagnosis	APACHE II	Vasoactive drug ($\mu\text{g.kg}^{-1}.\text{min}^{-1}$)	Outcome
1.	58	F	Pneumonia β -haemolytic streptococcal SS	27	NOR 0.05 DOP 3 DOB 15	S
2.	63	F	Pneumonia Cardiorespiratory arrest Septic shock	15	DOB 10	S
3.	68	M	Faecal peritonitis ARDS	22	DOB 3	S
4.	38	F	Guillain Barré SS, ARDS	12	NOR 0.05 ADR 0.06	NS
5.	73	M	Acute pancreatitis Perforated colon SS, ARDS	21	DOB 30 NOR 0.4	NS
6.	54	F	Pneumococcal pneumonia SS, ARDS	10	NOR 0.07	NS
7.	72	M	SS	28	DOB 15 NOR 0.28	NS
8.	57	M	Perforated diverticulum ARDS	3	DOB 5	S
9.	80	M	Subphrenic collection post- gastrectomy, SS, ARDS	25	DOB 4 NOR 0.12	NS
10.	52	F	Pneumococcal pneumonia ARDS	21	DOP 2 NOR 0.08	S
11.	65	F	SS, ARDS	21	DOB 5	NS
12.	64	F	SS (enterococci), ARF	25	DOB 15	NS
13.	45	M	Necrotising fasciitis, SS, ARDS	16	NOR 0.05	S

Clinical details: group 2.

14.	81	F	Faecal peritonitis Respiratory arrest, SS	18	ADR 0.04	NS
15.	59	F	Empyema, SS (β -haemolytic streptococci) Squamous carcinoma of lung	27	DOB 5 ADR 0.06	NS
16.	63	M	SS (β -haemolytic streptococci) Pneumonia Respiratory arrest	25	DOB 10 NOR 0.03	NS
17.	65	F	Necrotising fasciitis SS	20	DOB 12	S
18.	73	M	SS (coliforms) Indwelling urinary catheter	31	ADR 0.4 NOR 0.9	NS
19.	75	M	Faecal peritonitis Infarcted colon, SS	18	NOR 0.14	NS
20.	58	M	Ulcerative colitis Infarcted small bowel	21	NOR 2	NS
21.	80	M	Pneumonia SS	33	NOR 0.04 ADR 0.06	NS
22.	42	F	Post-colectomy SS	15	DOB 5 NOR 0.3	S
23.	70	M	Post-cystectomy (carcinoma bladder) SS	35	ADR 0.15 NOR 0.2	S

SS, septic shock; ARDS, adult respiratory distress syndrome; ARF, acute renal failure; DOP, dopamine; DOB, dobutamine; ADR, adrenaline; NOR, noradrenaline; S, survivor; NS, nonsurvivor.

dence of $\dot{V}\text{O}_2$ on oxygen delivery at levels of delivery which are well above the previously described critical level [13]. Oxygen consumption can thus be increased by increasing delivery, suggesting that tissue perfusion is inadequate. The mechanisms responsible for this phenomenon are not completely clear but probably involve a combination of factors including maldistribution of tissue blood flow due to

vaso-deregulation [21], an increase in capillary-to-cell diffusion distance caused by the interstitial oedema associated with capillary leak [22] and dysfunction of organelles as a result of the mediators and toxic elements generated and released in the inflammatory responses of sepsis and ARDS [23]. In addition, the demand for oxygen may be increased by fever, shivering or rigors [24]. Many investiga-

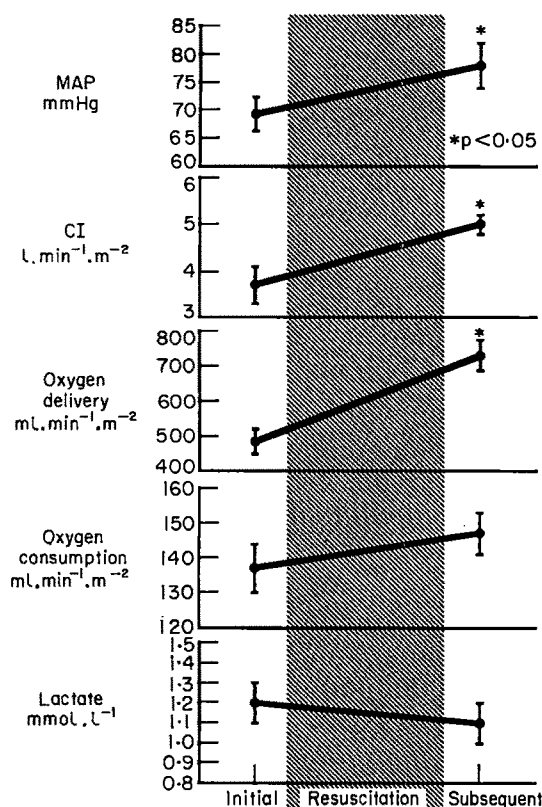


Fig. 1. Haemodynamic, oxygen transport and blood lactate changes during resuscitation in group 1 (initial blood lactate < 2 mmol.l⁻¹). MAP, mean arterial pressure; CI, cardiac index.

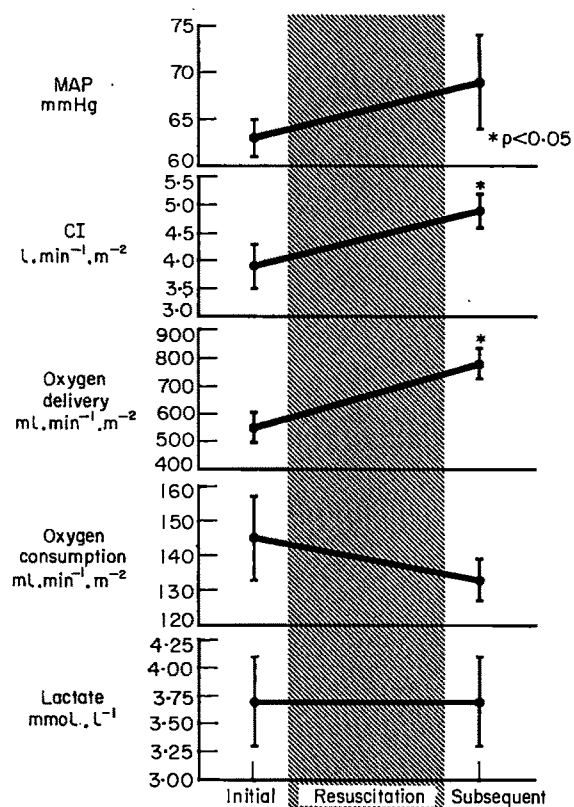


Fig. 2. Haemodynamic, oxygen transport and blood lactate changes during resuscitation in group 2 (initial blood lactate > 2 mmol.l⁻¹). MAP, mean arterial pressure; CI, cardiac index.

Table 2. Haemodynamic variables at baseline and after initial resuscitation. Data are presented as mean (SEM).

	Group 1 (Lactate < 2 mmol.l ⁻¹) n = 13		Group 2 (Lactate > 2 mmol.l ⁻¹) n = 10	
	Baseline	Resuscitation	Baseline	Resuscitation
HR (beat.min ⁻¹)	109 (6)	117 (5)	128 (5)†	127 (4)
MAP (mmHg)	70 (3)	78 (4)*	63 (2)	69 (5)
PAOP (mmHg)	13 (2)	15 (1)	13 (2)	13 (1)
CI (l.min ⁻¹ .m ⁻²)	3.7 (0.4)	5.0 (0.2)*	3.9 (0.4)	4.9 (0.3)*

MAP, mean arterial pressure; HR, heart rate; PAOP, pulmonary artery occlusion (wedge) pressure; CI, cardiac index; difference from baseline: *p < 0.05; between group comparison: †p < 0.05.

Table 3. Lactate and oxygen transport variables at baseline and after initial resuscitation. Data are presented as mean (SEM).

	Group 1 (Lactate < 2 mmol.l ⁻¹ ; n = 13)		Group 2 (Lactate > 2 mmol.l ⁻¹ ; n = 10)	
	Baseline	Resuscitation	Baseline	Resuscitation
Oxygen delivery (ml.min ⁻¹ .m ⁻²)	484 (36)	730 (44)**	549 (54)†	779 (54)*
Oxygen consumption (ml.min ⁻¹ .m ⁻²)	137 (7)	147 (6)	145 (12)	133 (6)
OER (%)	29 (2)	21 (1)*	28 (3)	18 (2)*
SvO ₂	69 (2)	77 (1)*	71 (3)	80 (2)*
Qs/Q _t (%)	28 (3)	32 (3)	27 (3)	34 (3)
Lactate (mmol.l ⁻¹)	1.2 (0.1)	1.1 (0.1)	3.7 (0.4)†	3.7 (0.4)

OER, oxygen extraction ratio; SvO₂, mixed venous oxyhaemoglobin saturation; Qs/Q_t, pulmonary shunt fraction; difference from baseline: *p < 0.05, **p < 0.005; between group comparison: †p < 0.05.

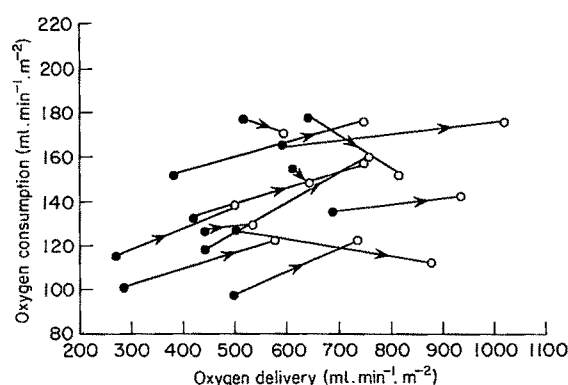


Fig. 3. Oxygen delivery and oxygen consumption changes during resuscitation in individual patients in group 1.

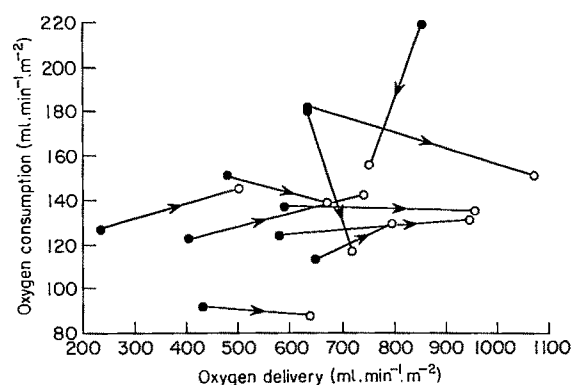


Fig. 4. Oxygen delivery and oxygen consumption changes during resuscitation in individual patients in group 2.

tors have confirmed this phenomenon [9–12, 14, 15]. In some of these studies, it has been suggested that $\dot{V}O_2$ rises significantly only in those individuals with severe tissue hypoxia, as evidenced by critically elevated blood lactate concentrations. This is in accordance with the study of Duff *et al.* [8] who showed that their septic patients with the highest lactate values had the lowest $\dot{V}O_2$ levels. There is, however, some disagreement about the value of hyperlactataemia as a predictor of supply-dependency in sepsis and ARDS. Vincent *et al.* [14] have used low-dose dobutamine ($5 \mu\text{g} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) to perform a short oxygen flux test in an attempt to disclose supply dependency of $\dot{V}O_2$ in stable patients with heart failure or sepsis. Dobutamine resulted in significant increases in CI and oxygen delivery but $\dot{V}O_2$ increased only in those patients with elevated blood lactate concentrations. Gilbert *et al.* [25] also demonstrated increased $\dot{V}O_2$ with increased oxygen delivery in response to infused catecholamines in septic patients with elevated lactate concentrations. However, previous work has suggested that the relationship between tissue hypoxia and hyperlactataemia in shock is much less clear-cut than may have been suggested [16]. Indeed, there is a subgroup of patients with the clinical and haemodynamic features of shock who have normal lactate concentrations.

We undertook the present study in the light of these controversies and because of the possibility that unrecognised tissue hypoxia may be present even in the absence of a critically elevated lactate concentration. We chose to include unstable patients during initial resuscitation in contrast to previous investigators who studied relatively stable, resuscitated patients. We were interested particularly in the relationship between oxygen delivery and consumption in patients without hyperlactataemia over this initial period. The timespan of study (up to 6 h, mean 3.2) was similar to that of previous work [15], although it might be suggested that the overall oxygen demand could change substantially over the study period described. This is a valid point, although work with continuous monitoring of mixed venous oxygen saturation and continuous on-line $\dot{V}O_2$ measurement shows that oxygen demand can change significantly from minute to minute in otherwise apparently stable patients [24, 26]. Unlike other investigators, we have rigorously standardised other factors which may affect $\dot{V}O_2$ or lactate independently. Patients with hyperglycaemia and liver failure were excluded. The contribution of work of breathing to $\dot{V}O_2$ and lactate production has been shown to

be considerable [27], and so all patients in our study were sedated and their lungs ventilated mechanically.

We allocated our patients to groups on the basis of blood lactate concentration rather than the type of resuscitation therapy received. Previous studies have concentrated on the use of either volume or vasoactive drugs. In both groups, significant increases of CI and oxygen delivery were achieved, but in the hyperlactataemic patients the increase in blood pressure achieved was not significant despite the enthusiastic administration of fluids and vasopressors. We have suggested previously that hyperlactataemia will be cleared reliably in the majority of shock patients only when flow, oxygen delivery and blood pressure are increased concurrently [16]. This hypothesis is supported by the results of those studies in which CI and oxygen delivery have been increased with no accompanying improvement in MAP from low levels [3]. In patients who remained hypotensive despite significantly increased blood flow, lactate concentrations remained high or increased further, with 100% mortality.

In our study, the inability to improve flow and MAP together in patients 14, 16, 18, 19 and 20 was associated with increasing lactate concentrations and death. The influence on the response to therapy of the underlying disease process, the inability to eradicate sepsis and pre-existing severe cardiorespiratory dysfunction have all been suggested as potential reasons for the poor outcome in these patients.

Examination of the relationship between oxygen delivery and consumption in individual patients reveals another tantalising discrepancy. The pooling of data for statistical analysis obscures individual idiosyncratic responses of $\dot{V}O_2$ to oxygen delivery. In particular, $\dot{V}O_2$ increased by between 17 and 35% in six patients with normal lactate concentrations. There were no increases in lactate, and this suggests that the increase in $\dot{V}O_2$ was not demand-led by a catecholamine-induced rise in metabolism. It is possible that unrecognised tissue hypoxia is present in critically ill patients even in the absence of critically elevated blood lactate. Since the production of hyperlactataemia in experimental models often requires combinations of severe anaemia, hypoxaemia, reduced cardiac output and profound hypotension, the finding of elevated lactate concentrations in clinical situations is likely to indicate an advanced degree of tissue hypoxia. The finding of normal lactate concentrations in patients with acute cardiorespiratory failure due to

sepsis and ARDS may not preclude the presence of a tissue oxygen debt. As blood lactate appears to be a fairly insensitive marker of tissue hypoxia, we are currently re-evaluating excess lactate, a potentially more sensitive guide [1]. Justifiable concerns on the effects of vasoconstrictors on regional blood flow and hence tissue oxygenation have been voiced, and recent work has documented the unpredictability of splanchnic blood flow responses, highlighting the need to withdraw vasoconstrictors as early as possible [28].

Basic therapy in these patients should therefore be directed to improving oxygen delivery and blood pressure concurrently, whilst attempting to correct or remove the underlying cause. The levels of oxygen delivery and MAP required in any individual situation remain very difficult to determine although a randomised study has shown an improvement in survival when supranormal oxygen delivery is achieved [29]. It appears clear that the finding of a normal blood lactate concentration should not encourage complacency, since this does not exclude supply dependency. Further research into the effects of independent factors such as sedation, paralysis and mechanical ventilation on the relationship between oxygen delivery and consumption is required to further unravel the complex pathophysiology.

Acknowledgment

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Cardiovascular effects of forces applied during laryngoscopy

The importance of tracheal intubation

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Summary

The relationship between the forces applied during laryngoscopy and cardiovascular changes were studied in patients undergoing laryngoscopy with or without intubation. This enabled us to differentiate between the cardiovascular effects of laryngoscopy and the effects of tracheal intubation. The forces applied during laryngoscopy were only weakly related to the cardiovascular changes, whereas tracheal intubation had a major influence. The many difficulties encountered in interpreting results from these studies are discussed. It is concluded that tracheal intubation causes more cardiovascular changes than laryngoscopy in routine uncomplicated procedures.

Key words

*Intubation, tracheal; complications.
Equipment; laryngoscope.*

Laryngoscopy and subsequent tracheal intubation cause cardiovascular changes which can lead to myocardial ischaemia and arrhythmias [1]. A number of factors influence the magnitude of these changes notably, induction agents [2,3], opioids [4, 5], neuromuscular blockers [6,7], as well as patient-dependent variables such as age [8] and hypertension [9,10]. Moreover, the type of blade used has been reported to affect cardiovascular changes [11].

Previously, attempts were made to differentiate between the cardiovascular effects of laryngoscopy and those of tracheal intubation. A much better understanding of the aetiology of these changes is needed in order to find ways of reducing them and to develop methods for prevention instead of, or in addition to, methods to block them pharmacologically.

A variety of studies suggest that the forces transmitted by the laryngoscope blade on to the base of the tongue are a major stimulus [12–15]. However, the results of these studies are often difficult to interpret and in some the laryngoscopic technique seemed far removed from common clinical practice.

Recently, a curved blade laryngoscope has been developed with which the forces applied during laryngoscopy can be measured [16]. Quantification of these forces permits their comparison with the cardiovascular responses to laryngoscopy [17], and introduces the possibility of distinguishing the cardiovascular effects of laryngoscopy from those of tracheal intubation.

The initial aim of this study was to investigate the effects

of the applied forces on cardiovascular parameters during routine laryngoscopy with tracheal intubation (group 1 patients). On the basis of the results of the first part of the study a second study was performed concentrating on cardiovascular responses to tracheal intubation as a stimulus itself (group 2 patients).

Patients and methods

Seventy-three patients, 49 in group 1 and 24 in group 2, scheduled for elective surgery, participated in the study. Approval from the Hospital Ethics Committee and informed consent from each subject were obtained. All patients were adults, in ASA grades 1 or 2, in whom no difficulty with intubation was anticipated. Patients in whom tracheal intubation proved difficult, or was not achieved at the first attempt, were not studied.

Before laryngoscopy, the patient's head was placed in the 'sniffing' position. Laryngoscopy in all patients was performed by the same person using the technique for curved blade laryngoscopes. In all patients, height (cm), weight (kg), sex, age and body mass index (kg.mg^{-2}) were recorded, as was the presence or absence of maxillary incisors.

The following laryngoscopic factors were measured using a modified curved blade laryngoscope [16]: (1) the duration of laryngoscopy (t_l), (2) the maximally applied force (F_{max}), (3) the mean force (F_{mean}) and (4) the integral of the forces with time ($F \cdot t$). The forces acting along the axis of the

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laryngoscope handle are measured by a strain gauge-based sensor positioned between the handle and the blade. The instrument was calibrated before each measurement by placing a weight of 2 kg (= 19.6 N) on the laryngoscope blade, whilst keeping the handle in the vertical position.

Each patient received midazolam (2.5 mg) and atropine (0.5 mg) intramuscularly 60 min before surgery. The systolic and diastolic blood pressures (SBP, DBP) and heart rate (HR) were recorded at 1 min intervals by a Dinamap 1846 SX automatic oscillometric blood pressure monitor with attached printer.

The timing of the cardiovascular measurements, pharmacological interventions and laryngoscopic procedures are shown in the flow diagram (Fig. 1). All cardiovascular measurements were carried out at 1 min intervals. The first three measurements were averaged to represent control readings. Directly after the last control measurements, fentanyl ($2 \mu\text{g.kg}^{-1}$) was administered. After the two subsequent cardiovascular measurements thiopentone ($4\text{--}5 \text{ mg.kg}^{-1}$) and suxamethonium ($1.0\text{--}1.5 \text{ mg.kg}^{-1}$) were given. Ventilation was manually assisted using a face mask and reservoir bag with 66% nitrous oxide in oxygen. Two minutes after the start of suxamethonium administration, directly following measurement PreL, the first laryngoscopic procedure was started, cardiovascular measurements continuing for 3 min ($t = 1$, $t = 2$, $t = 3$). In patients in group 1, laryngoscopy was directly followed by tracheal intubation. In group 2 patients, laryngoscopy was performed but the tip of the tube was positioned just above the laryngeal inlet avoiding direct contact (2 (a)). The tip was kept in this position for 3 s after which it was removed and ventilation was re-instituted with the face mask. Directly after the second cardiovascular measurement following the first laryngoscopy ($t = 2$), a second laryngoscopy was performed in group 2 patients and on this occasion was immediately followed by tracheal intubation (2 (b)).

Statistical methods

Linear regression analysis or Student's two sample *t*-test was used to study the univariate relationships between the

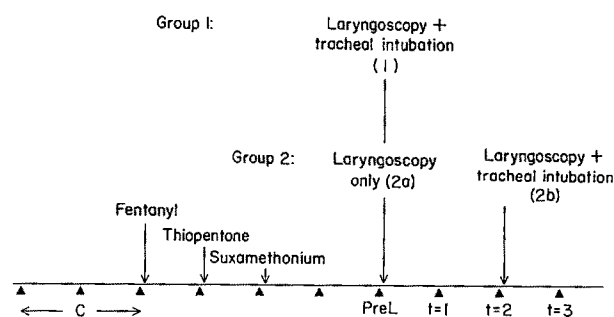


Fig. 1. Flow diagram of the investigational protocol. All cardiovascular measurements ($\blacktriangle$) were carried out at 1 min intervals. The first three measurements were averaged to represent control readings (C).

patient characteristics and the laryngoscopic measurements. The differences between the groups were compared by the Student's two-sample *t*-test. In order to obtain approximate normal distributions, t_1 and F^*t were logarithmically transformed. To determine whether the variability in a variable *Y* increased or decreased when *X* increased, the ordinary regression line of *Y* and *X* was determined initially. Subsequently, the absolute deviations of the *Y* values from the regression line were correlated with *X*. The level of significance was set at 5%.

Results

The demographic details, laryngoscopic factors and measurements, and cardiovascular values are shown in Tables 1, 2 and 3. Compared with group 2, patients in group 1 were heavier ($p = 0.007$), had longer duration of laryngoscopy than patients of group 2(a) ($p = 0.01$) and had a higher systolic blood pressure ($p = 0.003$).

The relationship between demographic details and cardiovascular values were studied in the group 1 patients. Age was not related to control heart rate or to changes in heart rate at 1 (Fig. 2), 2 or 3 min after laryngoscopy and intubation compared to control readings. However, age was significantly negatively related to variability of control

Table 1. Physical characteristics; values are mean (SD) and range, or ratios.

	Group 1		Group 2		p
Height; cm	174.0 (9.0)	155–197	169.0 (10.7)	149–190	0.06
Weight; kg	74.0 (13.9)	50–113	65.7 (10.5)	49–90	0.007
Age; years	38.0 (17.0)	16–75	36.1 (14.6)	15–78	0.55
Male:Female	22:27		9:15		0.55
with incisors					
without incisors	40:9		19:5		0.80

Table 2. Measured laryngoscopic values in groups 1, 2(a) and 2(b). Values are mean and (SD).

	Group 1		Group 2(a)		Group 2(b)		
		p value difference 1 v 2(a)		p value difference 2(a) v 2(b)		p value difference 2(b) v 1	
t ₁ (s)	16.3 (8.5)	0.01	12.5 (3.9)	0.26	13.5 (3.8)		0.66
F _{max} (N)	35.0 (12.0)	0.82	37.0 (11.0)	0.42	35.0 (12.0)		0.77
F _{mean} (N)	20.0 (6.0)	0.17	22.0 (7.0)	0.45	21.0 (6.0)		0.54
F* <i>t</i> (Ns)	324.0 (194.0)	0.31	285.0 (161.0)	0.90	279.0 (124.0)		0.20

t_1 , duration of laryngoscopy; $F_{\max}$, maximally applied force; F_{mean} , mean applied force; F^*t , force-time integral; group 1, laryngoscopy and intubation; group 2(a), laryngoscopy only; group 2(b), laryngoscopy and intubation.

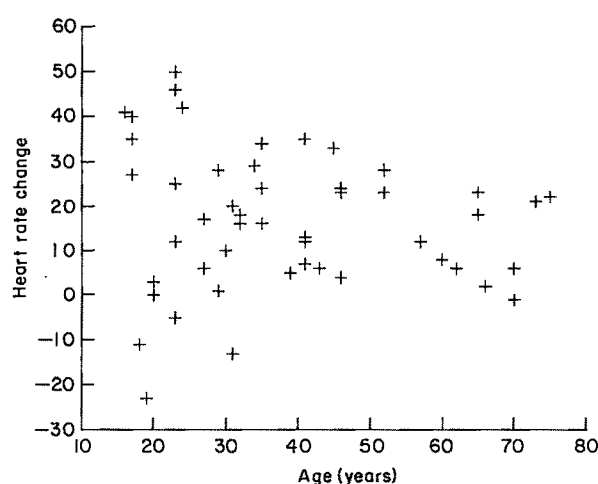


Fig. 2. Group 1: The effect of age (years) on changes in heart rate measured 1 min after the start of laryngoscopy with subsequent tracheal intubation compared to control values.

heart rate ($r = -0.46/p = 0.0008$) and changes in HR at 1 ($r = -0.42/p = 0.003$), 2 ($r = -0.33/p = 0.02$) and 3 min ($r = -0.43/p = 0.002$) after laryngoscopy. Control heart rates exceeding $90 \text{ beat} \cdot \text{min}^{-1}$ were only seen in patients aged under 55 years.

There was a significant relationship between age and control systolic ($r = 0.41/p < 0.005$) and diastolic ($r = 0.29/p < 0.01$) blood pressures, but age was not related to pressure changes at 1, 2 and 3 min compared to control readings. Height, weight, sex, body mass index and presence of maxillary incisors were not related to the cardiovascular values. Compared to control readings, there was no significant relationship between t_1 and changes in HR at 1 and 2 min after laryngoscopy, but t_1 was significantly related to changes in SBP at 2 min ($r = 0.27/p = 0.05$) and DBP at 1 and 2 min ($r = 0.34/p = 0.02$ and $r = 0.39/p = 0.006$ respectively).

There was no significant relationship between the forces applied during laryngoscopy and the cardiovascular results. There was only a trend for F_{max} to be related to changes in SBP at 2 min after laryngoscopy ($r = 0.25/p = 0.09$).

No significant relationship could be detected between $F \cdot t$ and changes in HR after laryngoscopy but $F \cdot t$ was significantly related to SBP at 2 min ($r = 0.3/p = 0.04$) and DBP at 1 ($r = 0.37/p = 0.01$) and 2 min ($r = 0.36/p = 0.01$) compared with control readings.

In group 1 patients, mean HR, SBP and DBP increased significantly after laryngoscopy and intubation ($p < 0.001$,

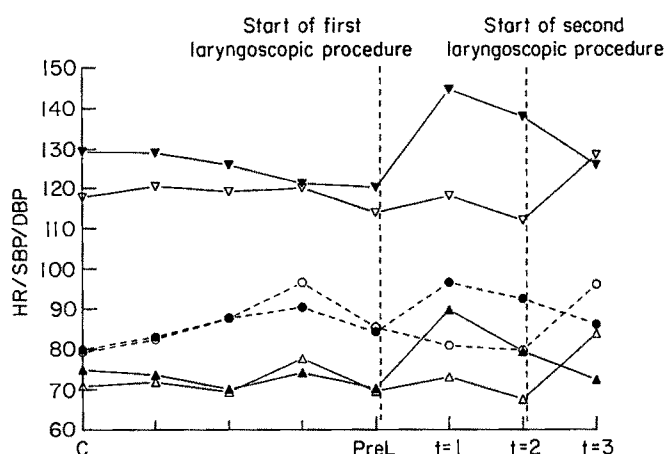


Fig. 3. Heart rate ($\bullet, \circ$), systolic blood pressure ($\blacktriangledown, \triangledown$) and diastolic blood pressure ($\blacktriangle, \triangle$) (groups 1 and 2, respectively) all measured at 1 min intervals. Control readings (C) were averaged over three measurements, readings at PreL were taken directly before start of first laryngoscopy; $t = 1$, $t = 2$ and $t = 3$: readings taken at 1, 2 and 3 min after the start of the first laryngoscopic procedure. The start of the first (groups 1 and 2) and second (group 2) laryngoscopy is indicated. For the exact sequence of events see Fig. 1

$p < 0.001$ and $p < 0.001$ respectively) (Fig. 3). In contrast, in group 2 patients there were no significant changes when laryngoscopy alone was performed, but when laryngoscopy and intubation were performed simultaneously, HR, SBP and DBP increased significantly at $t = 3$ (Fig. 1) ($p < 0.001$, $p = 0.01$ and $p < 0.001$ respectively). Moreover these changes were not significantly different from the changes after laryngoscopy and intubation in group 1.

Discussion

The cardiovascular responses to laryngoscopy and subsequent intubation are influenced by many factors, one of which is age. In contrast to a previous study in 27 patients [8], we could not detect a significant relationship between age and increased heart rate. However, the variability of HR changes decreased with increasing age, younger patients showing more extreme changes.

The results of the first part of this study suggest that the most important laryngoscopic factor influencing the cardiovascular response was duration of laryngoscopy, the forces applied having only minor effects. The latter was confirmed by the absence of a relationship between the

Table 3. Mean (SD) cardiovascular values in groups 1 and 2.

		C	PreL	t = 1	t = 2	t = 3
Heart rate ($\text{beat} \cdot \text{min}^{-1}$)	Group 1	80 (18)	84 (11)	97 (15)†	92 (14)	86 (14)
	Group 2	80 (14)	85 (12)	81 (9)‡	80 (12)	96 (15)§
Systolic blood pressure (mmHg)	Group 1	130 (17)*	120 (20)	145 (22)†	138 (26)	126 (24)
	Group 2	118 (13)	114 (20)	118 (21)‡	112 (19)	128 (24)§
Diastolic blood pressure (mmHg)	Group 1	75 (8)	70 (11)	90 (14)†	79 (14)	72 (12)
	Group 2	71 (9)	70 (12)	73 (14)‡	68 (13)	84 (19)§

*, significantly different compared to group 2; †, significantly different compared to values PreL; ‡, significantly different compared to group 1; §, significantly different compared to values $t = 2$; C, control readings; PreL, readings preceding first laryngoscopy; $t = 1, 2, 3$; measurements 1, 2 and 3 min after start of first laryngoscopy (see Fig. 1).

presence of maxillary incisors and cardiovascular changes. The presence of maxillary incisors has been shown to have a dominant effect on the forces needed [18]. We are unaware of studies that describe the influence of duration of routine laryngoscopy on heart rate and arterial blood pressure. The results of the second part of the study confirm that routine laryngoscopy as normally performed in clinical practice has only a minor influence on cardiovascular function. However, there are unexplained differences between groups 1 and 2 notably in weight and duration of laryngoscopy. Both of these had minimal effects on cardiovascular values in respect of laryngoscopy alone and we feel that they have no substantial influence on the main conclusions of this study.

A study relating forces applied during laryngoscopy to cardiovascular changes was published recently [19]. In this study 'impulse', the equivalent of $F \cdot t$, was found to be highly significantly related to the cardiovascular changes, tracheal intubation having additional effects. However, the mechanical characteristics of the instrument used have not been validated, the values for 'impulse' being extremely low compared to our values and those of other investigators [20]. Moreover, laryngoscopy was performed using a nonstandard technique.

Studies concerning the relative contribution of laryngoscopy and intubation to the cardiovascular changes are beset by problems. Firstly, laryngoscopy can be carried out easily without intubation, but it is more difficult to perform intubation in a comparable situation without laryngoscopy. Secondly, two stimuli administered within a short time interval in general do not result in a doubling of the response. More often there is a nonlinear, usually logarithmic, relationship between stimulus and response [21]. If laryngoscopy and tracheal intubation were stimuli of the same magnitude, intubation after laryngoscopy would lead to a relatively small additional increase in cardiovascular (or hormonal) response compared to the initial response to laryngoscopy alone. In this situation it might be erroneously concluded that laryngoscopy is a greater stimulus than intubation. Unfortunately, we are unaware of investigations specifically addressing this problem. Thirdly, a difficulty arises when two very powerful stimuli are involved, which can both lead to a (sub)maximal response. Again, in the situation described above the same erroneous conclusion might be drawn. A further problem concerns the effect the anaesthetic technique might have on the separate responses to laryngoscopy and intubation. Both the drugs and technique used must be standardised—but is there a fixed time allowed for laryngoscopy and/or a predetermined force applied or is laryngoscopy to be performed normally with just enough force to visualise the glottis and bring the tube into position? As duration of laryngoscopy is normally less than 30 s [20] the results of studies in which it takes longer than this have less clinical relevance. The final point concerns studies in which local anaesthetics have been applied. There is much controversy about the effectiveness of these agents in preventing cardiovascular changes [22]; some receptors are submucosal and are not blocked topically [23].

In this study the laryngoscopic measurements are related to the cardiovascular changes induced by laryngoscopy, assuming they are a measure of laryngoscopic stimulus strength. However, the forces applied by the laryngoscope are not homogeneously distributed along the blade. Forces

exerted by the tip of the blade might be more stimulating than the others. This could lead to confusion in the interpretation of the relationship between laryngoscopic measurements and the extent to which laryngoscopic stimulation affected the cardiovascular system. The results of this study suggest that in order to reduce cardiovascular changes, in addition to the methods already developed, attention should be focused on methods to reduce the stress of tracheal intubation; more effective local anaesthesia of the larynx and trachea and even more careful introduction of the tracheal tube. Moreover, the duration of laryngoscopy should be as short as possible. Obviously, our results do not rule out the possibility that cardiovascular changes can be caused by laryngoscopy alone.

In conclusion, however, tracheal intubation, more than laryngoscopy, causes cardiovascular changes in routine uncomplicated laryngoscopy and subsequent tracheal intubation in this group of patients with this type of induction technique.

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Cardiovascular effects of fiberoptic oral intubation

A comparison of a total intravenous and a balanced volatile technique

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Summary

The cardiovascular response to fiberoptic oral intubation under total intravenous anaesthesia with propofol or a balanced volatile technique with thiopentone/enflurane was compared in 50 patients of physical status ASA 1 and 2 who were scheduled for elective ear, nose and throat surgery. Patients were randomly assigned to receive propofol or enflurane. There was no significant difference between the two anaesthetic techniques in haemodynamic profile either before, during or after fiberoptic intubation (the study design was adequate to detect a 20% difference with > 90% statistical power), in incidence of postoperative sore throat or in time taken for intubation. In no patient did the oxygen saturation decrease to below 95% or the CO₂ tension exceed 5.8 kPa.

Key words

Equipment; fiberoptic bronchoscope.

Intubation, tracheal.

Anaesthetics, volatile; enflurane.

Anaesthetics, intravenous; propofol.

Awake fiberoptic intubation is the preferred technique for securing the airway in patients in whom difficult intubation is anticipated [1]. Fiberoptic intubation in the anaesthetised and fully relaxed patient is not yet a standard technique, but may be indicated for patients who have extensive dental reconstruction work, restricted head mobility or mouth opening and unsuspected difficult intubation. Teaching fiberoptic intubation skills in anaesthetised patients has also been advocated [2].

The cardiovascular responses to oral fiberoptic intubation in anaesthetised patients have previously been studied using volatile [3–6] and intravenous techniques [7]. A difference in severity of the stress response compared with orthodox intubation was demonstrated in the majority of studies using a volatile technique [3, 6], while no difference could be found in the study using total intravenous anaesthesia with propofol [7]. To our knowledge, there is no study comparing total intravenous anaesthesia and a balanced volatile technique for fiberoptic intubation. The stress response generated by conventional intubation is different for the two anaesthetic techniques. The present study was therefore designed to determine whether total intravenous anaesthesia with propofol would produce a clinically relevant difference in the fiberoptic haemodynamic intubation profile and postoperative patient

comfort when compared with a balanced volatile technique using thiopentone and enflurane.

Patients and methods

After approval by the local ethics committee and informed consent obtained, we studied 50 patients (ASA 1 and 2) undergoing elective ENT surgery requiring oral tracheal intubation. Patients taking antihypertensive medication and those with oesophageal reflux, morbid obesity or airway pathology were not studied.

The 50 patients were randomised into two groups: group P were given propofol and group E, enflurane. All patients were premedicated the night before with ranitidine 300 mg and 2 h before operation with midazolam 7.5 mg and atropine 1 mg orally. In the anaesthetic room, routine monitoring was started and a vein cannulated. Anaesthesia in group P was induced with fentanyl 0.2 mg (0.1 mg in patients older than 50 years) and lignocaine 20 mg for local intravenous anaesthesia, an infusion of propofol at a rate of 10 mg.kg⁻¹.h⁻¹ and a simultaneous bolus of propofol 1 mg.kg⁻¹. Anaesthesia in group E was induced with fentanyl 0.2 mg (0.1 mg in patients older than 50 years) and thiopentone 4 mg.kg⁻¹. As soon as adequate manual ventilation was established, atracurium 0.5 mg.kg⁻¹ was

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Table 1. Age, weight and sex characteristics (mean (SD) where applicable).

	Age (years)	Weight (kg)	Sex (M/F)
Propofol group <i>n</i> = 25	35.9 (14.3)	64.7 (14.0)	12/13
Enflurane group <i>n</i> = 24	31.7 (12.1)	69.9 (14.5)	14/10
Significance	NS	NS	NS

given intravenously to both groups. The patients' lungs were ventilated with 100% oxygen in group P and with 2% enflurane in oxygen in group E via a circle system using a fresh gas flow of 6 l.min⁻¹. Intubation was carried out when neuromuscular block was complete (no visible reaction to train-of-four stimulation of the ulnar nerve).

Systolic and diastolic arterial pressures and heart rates were measured using a noninvasive pressure monitor (Mark III BP-103 N with teleprinter) before induction of anaesthesia (baseline) and at 0, 1, 2, 3, 4 and 5 min after the start of fiberoptic intubation. A 20% difference was considered to be clinically relevant. Oxygen saturation was measured continuously, and end-tidal CO₂ was recorded before induction of anaesthesia and after intubation.

The induction-intubation interval and the duration of fiberoptic intubation (time between introduction of the fibroscope [Olympus BF 20 D] into the mouth until connection of the tracheal tube with the circle system) was recorded. Intubation with the bronchoscope was carried out by one of two experienced operators (H.G.S., S.C.M.) standing behind the patient's head. The tracheal tube was mounted on the bronchoscope and passed through the mouth, with sufficient jaw traction by an assistant to open the retropharyngeal space. When the tip of the broncho-

scope was positioned just above the carina, the tube was railroaded into the trachea and, after visual control of the correct placement of the tracheal tube, the bronchoscope was removed.

Statistical analysis was performed using paired and unpaired Student's *t*-test, Dunnett's multiple comparison tests and ANOVA as appropriate. *p* < 0.05 was considered significant.

Results

All patients were successfully intubated at the first attempt. Because of an incorrect dose of premedication, the data of one patient were not included in the results. Age, weight and sex (Table 1) were not significantly different for the two groups. No significant differences between the two techniques were found with regard to arterial saturation, end-tidal CO₂, induction-intubation intervals, time to successful intubation and incidence of sore throat (Table 2).

Details of the haemodynamic intubation profile for both groups are shown in Table 3. Dunnett's multiple comparison test did not result in any significant differences between the groups before, during and after intubation with respect to mean systolic and diastolic arterial pressure or mean heart rate. In both groups there was a significant decrease in systolic and diastolic arterial pressures after induction of anaesthesia (propofol 20% and 24%, respectively, vs enflurane 20% and 19%, respectively), with return to baseline values after intubation. Heart rate in the propofol group decreased significantly before intubation (14%) and increased to baseline values during and after intubation; in the enflurane group, heart rate remained stable after induction and increased significantly above control during and immediately after intubation (11%). Comparison of maximal haemodynamic mean values for systolic and diastolic

Table 2. Pre, intra- and postoperative parameters (Mean (SEM) where applicable).

Group	Before induction		Interval induction-intubation (min)	Duration of intubation (s)	After intubation		Incidence of sore throat (%)
	Oxygen saturation (%)	Pe'CO ₂ (kPa)			Oxygen saturation (%)	Pe'CO ₂ (kPa)	
Propofol <i>n</i> = 25	97.9 (1.6)	4.8 (0.6)	7.2 (1.7)	39.4 (15.1)	99.1 (1.1)	4.4 (0.3)	28
Enflurane <i>n</i> = 24	98.2 (1.1)	5.1 (0.5)	7.0 (1.8)	40.9 (17.3)	98.8 (0.9)	4.8 (0.6)	41
Significance	ns	ns	ns	ns	ns	ns	ns

Table 3. Mean (SD) systolic (SAP) and diastolic (DAP) arterial pressures and mean (SEM) values of heart rate (HR) in propofol and enflurane groups.

Group	Before induction	Before intubation	Min after start of intubation				
			1	2	3	4	5
Propofol <i>n</i> = 25							
SAP	128 (12)	103 (14)*	121 (22)	124 (19)	119 (18)	114 (16)	111 (12)
DAP	75 (10)	57 (9)*	72 (18)	72 (14)	69 (13)	65 (16)	63 (10)
HR	80 (18)	69 (13)*	79 (17)	78 (14)	78 (16)	74 (14)	75 (14)
Enflurane <i>n</i> = 24							
SAP	131 (13)	105 (10)*	129 (20)	134 (19)	129 (17)	126 (18)	122 (16)
DAP	74 (12)	60 (11)*	79 (18)	80 (16)	76 (14)	72 (12)	71 (15)
HR	74 (13)	75 (13)	87 (12)*	88 (12)	85 (12)	83 (13)	81 (13)

**p* < 0.05 compared with values before intubation.

arterial pressures (propofol 131/79, enflurane 139/84) as well as heart rate occurring during or after intubation (propofol 85, enflurane 92) revealed no significant difference between the groups.

Discussion

Both techniques produced a smooth haemodynamic profile with no significant differences between the groups in mean arterial pressure, heart rate or postoperative sore throat.

Although we did not measure end-tidal enflurane concentration or plasma propofol levels, both techniques produced a comparable degree of suppression of the haemodynamic reaction to fibreoptic intubation, achieved at 7 min after induction. In addition, maximum mean arterial pressure and heart rate were not significantly different between the groups confirming that both techniques were equivalent in obtunding the stress response. The heart rate profile was characteristic for the techniques used; a significant decrease in heart rate before and a return to baseline after intubation under propofol anaesthesia, and a stable heart rate after induction followed by a significant increase after intubation in the thiopentone/enflurane group. A specific central sympatholytic and/or vagotonic mechanism of propofol [8] might explain the nonsignificant differences. Because the study ensures a detection of differences of 20% with a statistical power of over 90%, we conclude that there is no clinical relevance to the differences between the two groups.

The findings in the enflurane group are in contrast with those reported by Finfer *et al.* [4] using a similar volatile balanced technique with enflurane in which intubation was associated with a significant increase in arterial pressure and heart rate when compared with baseline values before induction. Smith and colleagues [5], using isoflurane without fentanyl, also noted a significant increase in both arterial pressure and heart rate after intubation, but when fentanyl was added, the stress response was markedly attenuated [6]. A significant difference of the stress response to conventional intubation is reported in the studies comparing total intravenous anaesthesia and a balanced volatile technique, in spite of opioid medication [9, 10].

We suggest that the lack of a significant increase in arterial pressure in our study and an only moderate increase in mean heart rate (13 beat.min⁻¹ vs 25 beat.min⁻¹ in Smith *et al.*'s study [5] and 20 beat.min⁻¹ in Finfer's study [4]) after intubation in the enflurane group might be the result of several factors. First of all, we used a higher dose of fentanyl. Pretreatment with an opioid may partially obtund the haemodynamic response to laryngoscopy [6, 11, 12]. Secondly, a much longer period of mask ventilation before intubation and, associated with it, a deeper level of anaesthesia might have been of great importance in modifying the stress response [13]. Finally, the avoidance of aids to fibreoptic intubation helped to avoid stimulating sensitive laryngeal and hypopharyngeal structures [14].

In the propofol group, we confirmed our previous findings [7] showing a smooth haemodynamic intubation profile, with no significant differences between pre-induction and post-intubation haemodynamic data.

Whatever technique is chosen for fibreoptic intubation in anaesthetised patients, it should be kept in mind that the time to secure the airway is significantly longer than with

conventional laryngoscopy and that the railroading of the tracheal tube through the larynx over the fibreoptic is a blind manoeuvre. A diligent and unhurried approach is essential when using fibreoptic intubation in anaesthetised patients, all of whom should be at minimal risk of regurgitation.

We conclude that a balanced volatile technique can be used if fibreoptic intubation is achieved for ASA 1 and 2 patients within 60 s. However, if fibreoptic intubation proves difficult and several attempts are required to successfully place the tracheal tube (as in pathological airway anatomy or under teaching conditions) or the use of volatile agents is contraindicated (malignant hyperthermia or pregnant anaesthesia personnel), the use of propofol should seriously be considered. A stable plasma level of the anaesthetic agent is easily obtained by intravenous infusion schemes [15, 16] and despite the interruption of ventilation, an adequate depth of anaesthesia can be guaranteed for as long as necessary.

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Patient-controlled epidural analgesia following post-traumatic pelvic reconstruction

A comparison with continuous epidural analgesia

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Summary

A randomised, single-blinded study was conducted to compare patient-controlled epidural analgesia with continuous infusion epidural analgesia for the treatment of pain following post-traumatic pelvic reconstruction. The patient-controlled group ($n = 11$) received a background infusion of 4 ml.h^{-1} of bupivacaine 0.125% with fentanyl $1 \mu\text{g.ml}^{-1}$, and 3–6 ml bolus doses, self administered, as required (with a 15 min lockout interval). The continuous infusion group ($n = 12$) received a continuous infusion of the same solution through an identical apparatus, but with the demand button deactivated. This was started at 10 ml.h^{-1} and adjusted by the anaesthetist, as required, up to a maximum of 25 ml.h^{-1} . Pain scores, side effects, and the volumes of drug infused were recorded over the first 3 postoperative days. One patient from each group was withdrawn because of catheter-related problems. Pain scores were similar and the incidence of nausea and pruritus was low in both groups. There was no recorded instance of respiratory depression or hypotension and there was no significant difference between the groups in the volumes of drug solution received. Patient satisfaction was equally very good in both groups. Patient-controlled epidural analgesia is an effective means of providing pain relief after post-traumatic pelvic reconstruction, but did not significantly reduce analgesic requirements in comparison with continuous infusion epidural analgesia.

Key words

Anaesthetic techniques, regional; epidural.
Anaesthetics, local; bupivacaine.
Analgesics; fentanyl.
Equipment; patient-controlled analgesia.

Intravenous patient-controlled analgesia (PCA) has been used for over 20 years and its popularity is rapidly increasing. In the light of the recent report from the Royal College of Surgeons of England and College of Anaesthetists [1], this trend is likely to continue. The technique is associated with greater patient satisfaction [2], and often, reduced requirements for postoperative analgesia in comparison with conventional methods of pain relief. Epidural administration of local anaesthetics and/or opioids has also been shown to be highly efficacious in the relief of postoperative pain [3, 4]. Recently, a group from Sweden combined the concept of PCA with that of extradural opioids and studied postoperative patients who self-administered epidural morphine or pethidine [5]. Subsequently, a number of studies have evaluated the efficacy of patient-controlled epidural analgesia (PCEA) during labour, and have found it to be a safe and effective technique [6–10].

There have been a few preliminary reports describing the use of PCEA in nonlabouring patients [11–15], but only two have compared PCEA with continuous infusion epidural analgesia (CIEA) [16, 17]. The first of these compared the analgesic dosage requirement and side effect profile of hydromorphone, given by CIEA or PCEA, in patients undergoing a wide range of operations [16]. The other study compared the two methods to deliver fentanyl to patients who had undergone laparotomy [17]. In both studies, the PCEA group required considerably less drug to achieve a comparable level of analgesia.

Open reduction and internal fixation of major pelvic fractures requires extensive surgical exposure [18] and commonly results in severe postoperative pain. Our standard approach to the management of this pain is to use CIEA with a combination of fentanyl and bupivacaine. We wished to determine whether PCEA, with the same combination of drugs, would offer any advantages over CIEA

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in this group of patients. To address this issue we designed a randomised, single-blinded study to compare side effects, pain control, and drug requirements, between PCEA and CIEA in patients who had undergone post-traumatic pelvic reconstruction.

Methods

The study was approved by the institution's Committee on Human Volunteers Research and informed written consent was obtained from each patient. Twenty-three ASA 1 or 2 patients scheduled for post-traumatic pelvic reconstruction were entered into the study. Exclusion criteria included a Glasgow Coma Score of < 15, known hypersensitivity to any of the medications being used, coagulopathy, systemic infection, and pregnancy. Patients who were less than 18 or greater than 75 years old, or were due to have additional surgery, other than to the lower limbs, were also not studied.

On the evening before their operation, patients were shown how to use the PCA pump (Provider 5500, Pancreatec Inc., San Diego, USA), and how to score their pain on a visual analogue scale.

Patients were not premedicated. General anaesthesia was induced using thiopentone and fentanyl, and tracheal intubation was facilitated using suxamethonium or vecuronium. Anaesthesia was maintained, throughout the operation, by intermittent positive pressure ventilation with isoflurane (0.5–1.5%) in nitrous oxide and oxygen. Prior to surgery, the patient was turned into the lateral position and an epidural catheter was inserted at the L₂₋₃ or L₃₋₄ interspace. Following a test dose of 3 ml of 0.5% bupivacaine with 1:200 000 adrenaline, a further dose of 12 ml of 0.5% bupivacaine was given before surgery commenced. Additional epidural boluses of 5 ml 0.5% bupivacaine were given, depending on the haemodynamic response to surgery.

Upon arrival in the recovery room the patient was randomly assigned (sealed envelopes) to one of two groups. Patients in both groups received a loading dose of fentanyl 50 µg in 10 ml of bupivacaine 0.125% via the epidural catheter, which was then attached to the PCA pump. All patients subsequently received a solution of bupivacaine 0.125% containing fentanyl 1 µg.ml⁻¹, which had been prepared in bags of 500 ml by the hospital pharmacy. For patients in the PCEA group, the PCA pump was programmed to deliver a continuous infusion of 4 ml.h⁻¹ and a bolus dose of 3 ml on activation of the patient-demand button, with a lockout interval of 15 min. For patients in the CIEA group, the PCA pump was programmed to deliver a continuous infusion of 10 ml.h⁻¹ and the patient-demand button was deactivated (it still made a beeping noise when pushed). All patients were unaware to which group they had been assigned and received the same instructions to push the demand button whenever they required additional analgesia. Before each patient left the recovery area (usually about 2–3 h post-operatively), the quality of analgesia was scored on a 10 cm visual analogue pain scale and any side effects were recorded.

Patients were transferred to the high dependency ward. Nursing staff recorded the patient's heart rate, respiratory rate, blood pressure and level of consciousness every 2 h, and recorded the pain score on a visual analogue scale

every 4 h. At any time, if analgesia was deemed inadequate a member of the acute pain team reprogrammed the PCA pump; for the PCEA group, the bolus dose could be increased up to a maximum of 6 ml but the continuous infusion and lockout interval were kept constant; for the CIEA group, the infusion rate could be increased up to a maximum of 25 ml.h⁻¹. If patients in either group were pain-free for 4 consecutive hours, the dosage rates were reduced.

A member of the study group interviewed each patient at 24, 48 and 72 h postoperatively. The patient's assessment of their average level of pain over the previous 24 h was recorded on a 10 cm visual analogue scale. Additionally, patients were directly questioned regarding the incidence of pruritus, nausea and vomiting. At the 72 h assessment, patients were asked to record an overall impression of their pain control: very satisfactory, satisfactory, unsatisfactory, or very unsatisfactory.

Demographic data were analysed using Student's *t*-test, the volumes of drug solution used by *t*-test with the Bonferroni correction, and pain scores using the Wilcoxon rank sum test.

Results

Of the 23 patients studied, two (one from each group) had to be withdrawn. The patient withdrawn from the PCEA group initially had very good analgesia, but after 24 h he complained of severe pain from one side and demanded that the epidural catheter be removed. The other patient's epidural catheter became blocked after 36 h and despite having had excellent pain relief, he did not want another catheter inserted.

The patients' mean age, weight and blood loss are listed in Table 1. The higher mean weight in the CIEA group results from a greater proportion of men. The patients' age and average blood loss was not significantly different between the two groups.

At all assessment times, the median pain scores were

Table 1. Demographic data. Results are expressed as mean (SD).

	PCEA (n = 11)	CIEA (n = 12)	p*
Age; years	33 (14)	35 (8)	NS
Weight; kg	67 (7)	81 (14)	< 0.01
Blood loss; ml	1472 (1117)	1875 (1354)	NS
Number female	4	2	

*All analyses by *t*-test. NS, not significant.
PCEA, patient-controlled epidural analgesia;
CIEA, continuous infusion epidural analgesia.

Table 2. Visual analogue pain scores. Results are expressed as median (range)

	PCEA (n = 10)	CIEA (n = 11)	p*
Day 0**	2.0 (0.1–5.7)	1.4 (0–7.8)	NS
Day 1	2.6 (0.4–7.2)	1.4 (0.2–8.4)	NS
Day 2	1.5 (0–3.2)	2.1 (0.2–4.8)	NS
Day 3	1.3 (0–5.9)	1.1 (0–4.8)	NS

*All analyses by Wilcoxon rank sum test.

**Just before leaving the recovery area.

For abbreviations, see Table 1.

Table 3. Volumes (ml) of bupivacaine 0.125% with fentanyl $1 \mu\text{g.ml}^{-1}$ for the 1st, 2nd, and 3rd postoperative days. Results are expressed as mean (SEM).

	PCEA (n = 10)	CIEA (n = 11)	p*
Day 1	316 (34)	341 (41)	NS
Day 2	249 (33)	342 (53)	NS
Day 3	172 (24)	341 (53)	NS
Total	737 (72)	1003 (136)	NS

*All analyses by *t*-test with the Bonferroni correction.
For abbreviations, see Table 1.

similar in both groups (Table 2). At some time during the study period, four of the patients in the PCEA group required the maximum bolus dose of 6 ml, two required an increase to 5 ml, and the remainder were comfortable with 3 ml boluses. Three patients in the CIEA group required the maximum infusion rate of 25 ml.h^{-1} for at least part of the study period. Patients in the PCEA group required less of the drug solution than those in the CIEA group (Table 3), but this was not statistically significant when the Bonferroni correction for multiple comparisons was applied.

The incidence of side effects was similarly low in both groups (Table 4). The lowest recorded systolic blood pressure was 90 mmHg and no patient had a respiratory rate of less than $10 \text{ breath.min}^{-1}$. Throughout the study period, only one patient in the PCEA group and two in the CIEA group complained of pruritus, and these patients rated it as only mild. Three patients in the PCEA group and one in the CIEA vomited in the first 24 h period. During the second and third days no patients vomited, though between one and three from each group admitted to mild nausea.

All patients rated their overall pain control as satisfactory or very satisfactory, and in this respect there were no differences between the two groups (Table 5).

Discussion

This study has shown that PCEA with a solution of bupivacaine 0.125% with fentanyl $1 \mu\text{g.ml}^{-1}$ provides effective pain relief, equal to that provided by CIEA, in patients who have undergone post-traumatic pelvic reconstruction. Although there was a trend toward a reduced requirement for the bupivacaine/fentanyl solution in the PCEA group, this did not reach statistical significance. There was a low incidence of adverse effects. These were all minor and there were no differences between the two groups.

In our experience, the management of pain that follows

Table 5. Patients' overall rating of pain control.

	PCEA (n = 10)	CIEA (n = 11)
Very satisfactory	5	6
Satisfactory	5	5
Unsatisfactory	0	0
Very unsatisfactory	0	0

For abbreviations, see Table 1.

post-traumatic pelvic reconstruction can be a difficult problem. The operations usually involve extensive surgical exposure, often from a posterolateral approach [18], and leave the patient with considerable postoperative pain. Only a few studies have examined the use of PCEA outside of obstetric practice, but we hypothesised that the effectiveness of epidural analgesia, in combination with the flexibility offered by PCA, may provide optimal therapy for the patient who has undergone post-traumatic pelvic reconstruction.

Previous investigators have studied PCEA using either a mixture of bupivacaine and fentanyl [9, 10], or bupivacaine alone [6, 7]. We elected to use a combination of opioid and local anaesthetic because this has been shown to provide significantly better analgesia compared with local anaesthetic or opioid alone [19]. The ideal PCA drug would have a rapid onset of analgesic action, intermediate duration of action, and no side effects [20]. A mixture of fentanyl and bupivacaine provides more rapid pain relief than that provided by bupivacaine alone [21, 22]. Each patient in this study was to receive epidural analgesia for 3 days, so we were particularly keen to reduce side effects. We chose a relatively low concentration of bupivacaine (0.125%) and fentanyl ($1 \mu\text{g.ml}^{-1}$) in an attempt to minimise motor blockade and pruritus.

Although bolus volume and lockout interval may have an important bearing on the success of PCA with intravenous opioids [23], the precise influence of these factors on PCEA has yet to be determined. Investigators of previous PCEA studies have used: a 4 ml bolus of 0.125% bupivacaine with a 20 min lockout interval [6, 7], a 4 ml bolus of 0.125% bupivacaine with fentanyl $1 \mu\text{g.ml}^{-1}$ and a 10 min lockout [8]; a 3 ml bolus of 0.125% bupivacaine with fentanyl $2 \mu\text{g.ml}^{-1}$ and a 10 min lockout [10]; and a 4 ml bolus of the latter solution with a 15 min lockout [9].

The practice of adding a continuous infusion to supplement intravenous PCA with opioids is controversial. Some studies have shown better pain control and improved sleep if intravenous PCA is supplemented with a continuous infusion [24–26], while others have shown that the addition of a continuous infusion merely increases the dose of

Table 4. Numbers of patients experiencing side effects.

	Day 1		Day 2		Day 3	
	PCEA (n = 10)	CIEA (n = 11)	PCEA (n = 10)	CIEA (n = 11)	PCEA (n = 10)	CIEA (n = 11)
Nausea*	0	2	2	2	3	1
Vomiting	3	1	0	0	0	0
Pruritus	1	2	1	2	1	2

*Excluding those patients who vomited.
For abbreviations, see Table 1.

analgesic given without improving the pain control [27, 28]. However, the results of PCA using intravenous opioids should not be extrapolated to PCEA using a mixture of local anaesthetic and opioid. One study that has examined the effect of adding a basal infusion to PCEA failed to demonstrate any significant benefit, but this was using only hydromorphone [14].

We were concerned about possible difficulties in re-establishing adequate analgesia if the local anaesthetic block were to recede completely and therefore chose to add a basal continuous infusion of 4 ml.h^{-1} which remained fixed for the duration of the study. In an attempt to increase the flexibility of the PCEA technique, we adjusted the volume of the bolus dose between 3 and 6 ml, while fixing the lockout interval to 15 min. We considered that a lockout interval any shorter than this would not allow enough time to establish analgesia via the epidural route. Six patients in the PCEA group required an adjustment of their bolus dose to 5 or 6 ml, suggesting that the optimal bolus volume of this particular solution in this group of patients is 5 ml. The Provider 5500 infusion pump used in this study has a number of features that make it ideal for use in PCEA [29]. It delivers accurately in both infusion and bolus modes. It has an occlusion alarm pressure (1200–1360 mmHg) which will accommodate the high pressures generated during bolus dosing through the resistance of the epidural filter and catheter [29]. In addition, the drug reservoir which connects to the pump's dedicated infusion set, can be any intravenous fluid container. This enabled us to use 500 ml bags, providing enough drug solution for 24 h.

Previous studies demonstrating significant advantages of PCEA over CIEA used continuous infusions at a fixed rate in the CIEA group [7, 10, 16]. In contrast, patients in our CIEA group had their infusions adjusted according to their pain scores. In common with our study, that by Lysak *et al.* [8] failed to show a clear advantage for PCEA. However, in the latter study the infusion rate in the CIEA group was adjusted hourly and it is likely that such close medical attendance, accompanied by very frequent changes in infusion rates, merely duplicates the on-demand nature of PCEA [8, 10]. This assumption should not be applied to our study because the minimum time between changes in the rate of infusion was rarely less than 4 h.

We have analysed and presented only those pain scores collected by members of the study group. The patients' assessment of their pain over the previous 24 h was recorded as a single retrospective measurement. In comparison with repeated contemporaneous pain scoring, the reliability of this method of assessing pain has been questioned [30]. Although the nursing staff were doing four-hourly assessments of pain, their data was incompletely recorded and therefore unreliable for the purposes of this study.

Our patient groups were small in number. However, to standardise the two groups we elected to limit the study to those patients undergoing post-traumatic pelvic reconstruction. This is a relatively uncommon operation, even in major trauma centres, and it required 18 months to enroll 23 patients.

The incidence of side effects was similar in both groups. Throughout the 3 day study period, only three patients (one from the PCEA group and two from the CIEA group) complained of mild pruritus, and none required treatment.

This finding concurs with that from a previous PCEA study which found a 6% incidence of pruritus in patients receiving bupivacaine 0.125% with fentanyl $1 \mu\text{g.ml}^{-1}$ [8]. Considerably higher doses of epidural fentanyl are sometimes used in postoperative patients but this significantly increases the incidence of pruritus. In one study, 38% of patients receiving an epidural infusion of fentanyl at a mean dose of $1.52 \mu\text{g.kg}^{-1}.\text{h}^{-1}$ required diphenhydramine for pruritus [31]. Such an incidence of pruritus would be unacceptable in the setting of this study where the patients received epidural fentanyl for at least 3 days.

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Continuous extradural infusion of lignocaine 0.75% vs bupivacaine 0.125% in primiparae: quality of analgesia and influence on labour

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Summary

We studied 86 primiparous women with uncomplicated pregnancy and labour requesting extradural analgesia in labour. All the women were over 36 weeks of gestation with a cephalic-presenting singleton fetus. The women were allocated randomly to two groups: group A, who received an extradural infusion of lignocaine 0.75%, after an initial dose of 10 ml of lignocaine 1.5%, and group B, who received an infusion of bupivacaine 0.125% after an initial dose of 10 ml of bupivacaine 0.25%. All the women had their labour actively managed. Assessment of analgesia during labour and delivery, and the requirements for additional top-ups were noted, as were mode of delivery, requirement for oxytocic augmentation and incidence of fetal distress. Maternal and umbilical cord plasma concentrations of lignocaine were measured at delivery in 12 women receiving extradural lignocaine. There were no statistically significant differences between the two groups in terms of the mode of delivery, incidence of fetal distress, fetal heart rate abnormalities, or Apgar scores of the babies. Women in the bupivacaine group had a significantly better quality of analgesia during both the first and second stages of labour ($p = 0.0005$) and required fewer top-ups than those in the lignocaine group. However, the requirement for oxytocin augmentation during the first and second stages of labour was significantly less in the lignocaine group ($p = 0.004$). Similarly, the duration of the second stage was shorter compared with the bupivacaine group. In spite of high plasma concentrations of lignocaine, no side effects were noted in either mothers or babies.

Key words

Anaesthesia; obstetric.

Anaesthetic techniques, regional; extradural.

Anaesthetics, local; bupivacaine, lignocaine.

Extradural infusion of a low concentration of local anaesthetic is becoming an acceptable alternative to intermittent boluses as a method of pain relief during labour. Benefits are said to include stable continuous analgesia with minimal motor blockade, a reduced level of systemic toxicity and a decreased incidence of hypotensive episodes. Also, should the catheter migrate into the subarachnoid space, a total spinal blockade would be less likely with this method of administration [1].

We felt that lignocaine would merit re-investigation, in the context of infusion techniques, because of its rapid onset and shorter duration of action. Although lignocaine has a higher fetal : maternal ratio than bupivacaine, work by Abboud and co-workers [2] showed a higher incidence of fetal heart rate abnormalities in mothers who had been given bupivacaine. They also suggested that bupivacaine

may be associated with a longer second stage of labour than lignocaine.

The present study was designed to evaluate, in a group of primiparae undergoing a uniform obstetric management, the effect of a continuous infusion of lignocaine compared with bupivacaine on the quality of analgesia, degree of motor blockade, outcome of labour and the effect on the neonate.

Method

The study was approved by the local ethics committee and verbal consent was obtained from all patients.

All women were healthy primiparae, with an uncomplicated pregnancy and requesting extradural analgesia. Patients with medical disorders (pre-eclampsia, diabetes,

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intra-uterine growth retardation) were not studied. All women were at or near term (36–42 weeks of gestation) with a live singleton fetus in cephalic presentation.

Extradural analgesia

Fetal heart rate monitoring and intravenous access were established. When the cervix was 3–5 cm dilated, an extradural catheter was inserted at the L₂₋₃ or L₃₋₄ interspace and a test dose of 3 ml lignocaine 2% administered to all the mothers. Those in group A then received 10 ml of lignocaine 1.5%, while those in group B received 10 ml of bupivacaine 0.25%. Once a sensory block from T₈ to S₅ was achieved, a continuous extradural infusion was started. Group A received lignocaine 0.75% whilst group B bupivacaine 0.125%. The rate of the infusion was set at 10 ml.h⁻¹ and additional top-up doses of 5 ml of the initial solution were given if analgesia was inadequate. The number of additional top-ups required was noted. The sensory block was assessed by loss of sensation to cold and the degree of motor blockade was monitored and graded according to the Bromage scale (none, partial, almost complete, complete) [3]. The infusions were continued until delivery of the baby. If a decision to proceed to instrumental or operative delivery was made, lignocaine 2% with adrenaline 1:200 000 was given to intensify the block. Maternal heart rate and systolic blood pressure were recorded every 5 min for the first 20 min following top-ups and thereafter every 30 min. Hypotension (systolic blood pressure < 85 mmHg), if observed, was treated promptly with the intravenous administration of ephedrine 6–10 mg, and compound sodium lactate solution.

All patients were seen on the following day by an anaesthetist unaware of the study and were questioned about the quality of analgesia during the first stage (excellent, good, fair and poor) and second stage (good, fair and poor) of labour.

Obstetric management

All women studied were managed according to the uniform policy for the active management of labour introduced in this hospital in 1985 [4]. Diagnosis of labour was made on strict criteria and vaginal examination performed 2-hourly to assess progress. Induction of labour, when needed, was achieved by a prostaglandin pessary inserted vaginally. Progress of labour was plotted on a straight line partogram which assumed that cervical dilatation would proceed at 1 cm.h⁻¹. Failure to progress in the first stage or the fetal head to descend in the second stage was treated with intravenous oxytocin. Women were not encouraged to bear down before the fetal head was visible.

Instrumental or operative delivery took place when there was fetal distress or failure to progress; the obstetrician was unaware of the solution used for extradural analgesia. Requirement for oxytocin, duration of the second stage of labour and the subsequent mode of delivery were noted. Intra-uterine fetal wellbeing was assessed by the fetal heart rate. Abnormalities such as type 2 decelerations, and their relationship, if any, to the use of the extradural top-up or infusion were recorded.

Venous umbilical cord and maternal blood samples were taken at delivery in 12 mothers in the lignocaine group. The samples were immediately centrifuged, and the serum was

stored at –20°C. The serum lignocaine was assayed by high pressure liquid chromatography. No interfering peaks were seen. The two major metabolites, monoethylglycinexylidide and glycinexylidide, were eluted well before the lignocaine and internal standard peaks. A calibration line was run over the concentration range 0.0 to 3.0 µg.ml⁻¹. The patient samples were all run on the same day with the standards and patient samples randomised. A set of quality control samples were placed every 10 samples/standards. The correlation coefficient of the calibration line was 0.9995 with a coefficient of variation for the quality control samples not exceeding 2.9%.

Infant condition (Apgar score) at 1 and 5 min after birth was evaluated by a paediatrician unaware of the local anaesthetic administered. Neonatal assessment of body tone, sucking and feeding was performed at birth and 24 h later.

Data are presented as means and range or standard deviations. Statistical analysis was performed using Mann–Whitney *U*-test, Chi-squared or Fisher exact test as appropriate. A value of *p* < 0.05 was considered statistically significant.

Results

There were no significant differences between the two groups in maternal height, weight, gestation and birth weight of the infants (Table 1). Seven women in the lignocaine group and six in the bupivacaine group received prostaglandin pessaries for induction of labour. The mean (range) duration of the extradural infusions was 4.55 h (1.3–9.8 h) for bupivacaine and 3.72 h (1.5–7.6 h) for lignocaine. No maternal hypotension (systolic blood pressure less than 85 mmHg) occurred in either group throughout the study period.

Thirty-three patients in the lignocaine group (78%) required more than one top-up, and many of these were receiving top-ups almost every hour. Conversely, 20 patients in group B (45%) required only one additional top-up. There was a statistically significant difference in the total numbers of top-ups between the two groups (*p* = 0.002).

Table 2 shows the patients' assessment of the quality of analgesia for the two drugs, with bupivacaine giving a significantly better quality of analgesia during both the first (*p* = 0.0005) and second (*p* = 0.03) stages of labour. Seven patients in the bupivacaine group and one in the lignocaine group required a general anaesthetic for Caesarean section and no analgesia assessment of the

Table 1. Patients' data, mean (range).

	Lignocaine (<i>n</i> = 42)	Bupivacaine (<i>n</i> = 44)
Maternal age; years	25 (14–37)	26 (18–37)
Race;		
Caucasian	33	31
Noncaucasian	9	13
Weight; kg	72 (52–94)	74 (54–111)
Height; cm	161 (149–174)	161 (151–173)
Gestational age; weeks	40 (36–42)	40 (36–42)
Infant birth weight; kg	3.4 (2.3–4.9)	3.3 (2.2–4.2)

Noncaucasian = Asian, Indo-Pakistani, African.

Table 2. Quality of analgesia during the first and second stages of labour, and motor blockade.

	Lignocaine (n = 42)	Bupivacaine (n = 44)	p
First stage;			
Excellent	6	16	0.0005
Good	17	23	
Fair	17	5	
Poor	2	0	NS
Second stage;			
Good	23	27	0.03
Fair	4	9	
Poor	14	1	
Motor blockade;			
None	38	37	NS
Partial	4	7	NS
Almost complete	0	0	
Complete	0	0	

second stage was reported. The degree of motor blockade was similar in both groups.

There was no statistically significant difference between the two groups in terms of spontaneous and instrumental mode of delivery (Table 3). The incidence of Caesarean section was greater in the bupivacaine compared with the lignocaine group, but this difference was not statistically significant. The indications for instrumental and operative deliveries were similar in both groups. More patients in group B required oxytocin, during either the first or the second stage of labour. However, as Table 3 shows, the duration of the second stage of labour in those women who had either spontaneous or instrumental vaginal delivery was significantly longer in the bupivacaine group ($p < 0.002$) than in the lignocaine group.

Following the initial extradural injection of local anaesthetic, late heart rate deceleration patterns occurred in 26 fetuses in the lignocaine group and 28 in the bupivacaine group. There were no significant changes in the baseline fetal heart rate during the infusions of both local anaesthetics. Apgar scores were above 8 in all but four cases in group A and three in group B, and there were no signifi-

cant changes in body tone, sucking and feeding patterns in either group after birth. Table 4 shows the total dose given and plasma maternal and fetal concentrations of lignocaine in the 12 mothers studied. Although some patients in this group received large doses of lignocaine, only one patient had a plasma concentration of lignocaine in the toxic range ($> 5 \mu\text{g}\cdot\text{ml}^{-1}$) at delivery (patient number 6). In spite of this high concentration, no clinical effects in the infant at birth and during the first 24 h were noted. None of the mothers in this group experienced symptoms of toxicity. The cord concentration did not rise to toxic levels, and the mean fetal : maternal ratio for lignocaine was 0.57.

Discussion

The less satisfactory quality of analgesia obtained with lignocaine in our study is not universally reported. Zador *et al.* [5] studied 22 women of mixed parity who were given 0.4% lignocaine, with or without adrenaline, as an extradural infusion in labour. Additional boluses were given for the second stage of labour to those women who received a plain lignocaine infusion. The authors commented that while additional doses were required for the second stage in some patients who received plain lignocaine, the overall quality of analgesia was good. Nevertheless, the need for additional top-ups would imply that analgesia was not as good as it could have been. Chestnut *et al.* [6] gave lignocaine as an extradural infusion during the first stage of labour to 53 primiparous women. For the second stage, normal saline was given to half the women whilst an additional top-up of lignocaine was given to the others. Pain relief during the first stage was reported to be good, and there was no statistically significant difference between the two groups in the quality of analgesia during the second stage. Abboud *et al.* [2] studied 61 women of mixed parity and found that when lignocaine was compared with bupivacaine and chloroprocaine, there was no difference in the need for top-ups. Therefore, the evidence for the efficacy of lignocaine is conflicting. However, the major criticism of these studies is that they used relatively small numbers of patients of mixed parity with no standardisation of the

Table 3. Conduct of labour and delivery.

	Lignocaine (n = 42)	Bupivacaine (n = 44)	p
Mode of delivery			
(a) Spontaneous vaginal	21 (51%)	20 (45%)	NS
(b) Instrumental			
Low forceps	16 (38%)	14 (33%)	NS
Rotational forceps	1 (2%)	1 (2%)	NS
Indication			
Failure to progress	7	6	
Fetal distress	10	9	
(c) Caesarean section	4 (9%)	9 (20%)	0.23
Indication			
Failure to progress	1	2	
Fetal distress	3	7	0.31
Position of vertex before delivery			
Occiput anterior	38	40	
Occiput posterior	2	3	
Occiput transverse	2	1	
Oxytocin augmentation	18 (43%)	33 (75%)	0.004
Duration of second stage; h	0.58 (0.13–2.45)	1.51 (0.26–2.62)	0.002

Table 4. Maternal and fetal blood concentrations of lignocaine in 12 patients.

Patient	Total dose; mg	Dose; mg.kg ⁻¹ .h ⁻¹	Maternal plasma concentration; µg.ml ⁻¹	Umbilical cord concentration; µg.ml ⁻¹	F:M ratio
1	450	1.58	3.35	1.58	0.47
2	650	6.77	2.74	1.49	0.54
3	870	3.89	4.39	2.45	0.56
4	762	3.22	2.57	1.03	0.40
5	853.1	2.19	1.27	1.12	0.88
6	938.7	3.0	5.63	1.61	0.29
7	945	2.38	3.51	2.45	0.70
8	1218	2.4	3.72	3.14	0.84
9	521.2	2.8	2.4	1.19	0.5
10	892.5	3.23	2.65	1.51	0.57
11	869	3.18	2.39	0.91	0.38
12	237	3.37	1.39	0.98	0.71
Mean	767.2	3.16	3.0	1.44	0.57
SD	263.6	1.52	1.22	0.63	0.18

F:M = fetal:maternal.

obstetric management. In this investigation we have attempted to study a reasonable number of primiparae receiving uniform obstetric management.

Bupivacaine has superseded lignocaine for use in extradural analgesia during labour because of its longer duration of action and lack of tachyphylaxis. Lignocaine was also associated with the syndrome of 'floppy but alert' baby as described by Scanlon in 1974 [7]. However, subsequent work undertaken to evaluate the neuro-behavioural effects on the fetus in a group of mothers having extradural lignocaine, as compared to a group of mothers receiving no pain relief during labour and delivery, did not show any major difference in neonatal outcome [8, 9]. Further work comparing the effect of extradural analgesia with bupivacaine, 2-chloroprocaine and lignocaine on the neonatal behavioural function did not show any difference between the three local anaesthetics [10]. However, the administration of 0.5% bupivacaine was associated with a higher incidence of fetal heart abnormalities compared with lignocaine. In our study no prolonged decelerations were observed during the period of extradural infusion in the two groups, and this might be explained by the fact that, at variance with the previous workers, we used a continuous infusion of local anaesthetic which minimises the high concentration effect across the placenta and does not cause maternal hypotension. The latter complication was reported by those authors in 10% of cases following a top-up with bupivacaine [10] and in 5% following lignocaine.

The mean plasma concentration of lignocaine measured at delivery in 12 mothers of group A (3.0 µg.ml) was greater, but not significantly different, from that reported by Abboud [2] and did not correlate with total dose of lignocaine administered. However, if we had measured the concentration after the administration of added top-ups, a higher plasma concentration may have been found [11]. Despite the high plasma concentration of lignocaine found in one mother, no signs of toxicity were observed and neonatal outcome was invariably good.

In our study the incidence of spontaneous vaginal delivery following lignocaine infusion was greater, but not significantly so, from that observed with bupivacaine. In the latter group there was also a significant increase in the

requirement for oxytocin augmentation during either the first or second stage of labour. The second stage of labour was significantly longer in the bupivacaine group than in the lignocaine group. These observations are in agreement with those reported by previous workers when either top-up or continuous infusion of lignocaine were used [2, 10]. A possible explanation for the shorter second stage of labour and decreased need for oxytocin augmentation observed with the lignocaine group might be the minimal interference of this anaesthetic agent on uterine activity [12] and descent of the baby to the pelvic floor.

In conclusion, continuous extradural infusion of 0.75% lignocaine in primiparae did not provide adequate analgesia during labour and delivery. However, it was associated with a low incidence of motor block, shorter duration of second stage, high incidence of spontaneous vaginal delivery and no detrimental effect on the neonate. It remains to be established whether a pharmacological manipulation of this infusion, e.g. addition of either an opioid or adrenaline, might decrease its requirement, provide satisfactory analgesia for delivery and maintain a reasonably low incidence of instrumental and operative deliveries.

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An assessment of an alternative method of internal jugular vein catheterisation

I. P. LATTO, J. A. HUGHES AND R. J. FALCONER

Summary

This study assessed a tried, but previously unpublished, method of internal jugular vein cannulation in 50 patients scheduled for elective cardiac surgery. The method involves using two landmarks: the larynx (thyroid and cricoid cartilages) together with the carotid artery (if it is palpable). The cricoid and thyroid cartilages were readily identifiable in 48 (96%) patients. The carotid artery was palpable in 45 (90%) patients, although some difficulty in palpation was recorded in 24 (48%) patients. There was a 98% success rate of venous cannulation. In 90% of cases the 21 gauge seeker needle entered the vein on the first or second attempt, and in 82% of cases the subsequent 18 gauge introducing needle entered the vein on the first attempt.

Key words

Anatomy; internal jugular vein, cricoid/thyroid cartilages, carotid artery.

There are many methods for cannulating the internal jugular vein [1], but some may not always be appropriate. Many techniques rely on identifying the sternomastoid muscle, whilst a few rely on identifying either the carotid artery or the internal jugular vein. The sternomastoid may be difficult to define in patients who are obese or have short, 'bull' necks. The carotid artery may not always be easily palpable and the internal jugular vein may be extremely difficult to palpate in a significant proportion of patients.

The purpose of our study was to evaluate a technique which primarily uses as a landmark the lateral border of the larynx together with the carotid artery, if it is palpable. It has been observed that many trainees fail to cannulate the internal jugular vein using the carotid artery as their principal landmark because they direct the needle too far laterally in relation to the artery. This may be from fear of puncturing the artery or because the artery is difficult to palpate. Coincidentally, whilst attempting cannulation, it was noted that when the pads of the operator's fingers rested on the lateral border of the larynx then the tips of the fingers in turn usually rested along the line of the carotid artery. With the fingers in this position the vein was successfully cannulated on a number of occasions when the artery was impalpable. It was decided to examine this method in a formal study.

Method

Approval for the study, on 50 patients, scheduled for elective cardiac surgery, was given by the District Research Ethics Committee. The trainee anaesthetists (operators), who were to cannulate the internal jugular vein, were given an instruction sheet. The pillow was removed, the patient positioned in a 15° Trendelenburg tilt with the neck extended approximately 10°. The operator was asked to locate and mark the cricoid cartilage and the top of the thyroid cartilage and draw a transverse line on the right side of the neck halfway between these two points. An indelible marker pen was used so that the mark would not be dissolved by antiseptic cleansing fluid. The middle three fingers of the operator's left hand were then positioned such that the pads of the fingers lay on the lateral aspect of the larynx with the finger tips pointing directly posterior i.e. at right angles to the coronal plane (Fig. 1). The operator was asked the following questions: (1) does the patient have a short, or 'bull' neck? (2) are the cricoid and thyroid cartilages easy to feel? (3) with the head at 45° to the left is the carotid artery (a) impalpable, (b) just palpable, (c) easily palpable, (d) bounding? (4) with the head at 45° to the left and the fingers placed as described, is the carotid artery (if palpable) (a) directly beneath the fingers, (b) up to one finger-breadth lateral, (c) more than one finger-breadth lateral?

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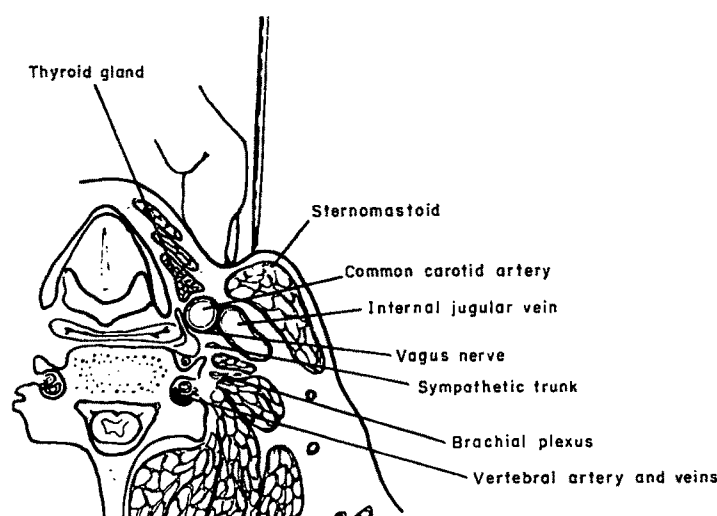


Fig. 1. Cross-sectional anatomy of the neck. The pads of the fingers lie on the lateral aspect of the larynx with the fingertips pointing directly posterior. (Reproduced, with permission from [3]).

In each case the study supervisor was asked to make independent observations and complete a form with an identical list of questions.

The operator then donned gown and gloves, before draping the patient prior to attempting cannulation. If, with the fingers placed as described, the tips of the operator's fingers had lain along the line of the artery then this position was re-adopted. If the artery had been palpable lateral to the operator's fingers then the fingers were positioned more laterally. After infiltration with lignocaine 1%, a 21 gauge seeker needle was inserted immediately lateral to the fingers at the level of the marked line. The needle was directed parallel to the sagittal plane and approximately 30–40° to the coronal plane. If the vein was not found after insertion of the seeker needle in this direction, the needle was slowly withdrawn to just beneath the skin and redirected in a more lateral direction (15, 30, 45, 60, 75°). Once the vein was located the seeker needle was left in place and used as a guide for the subsequent insertion of the 18 gauge introducing needle, the fingers of the left hand remaining on the larynx. Once the vein was successfully located with the introducing needle a 'Seldinger' catheter-over-wire technique was employed to complete cannulation. A maximum of six attempts were allowed with the seeker needle and three attempts with the subsequent introducing needle before the supervisor took over the cannulation attempt. The trainee was then asked the following two questions: (a) do you have any comments on this technique? (b) would you consider using this technique in future and, if so, under what circumstances?

Results

Data were obtained on 50 patients (39 males, 11 females) whose age ranged from 44 to 83 years, (mean age 64 years).

The cricoid and thyroid cartilages were easily palpable in 48 (96%) of the patients studied. It was noted that a 'bull' neck was present in the two patients in whom the cartilages were difficult to identify. In these cases both the operator and supervisor could identify the larynx but experienced difficulty in differentiating between the laryngeal cartilages. The results regarding the relative ease of palpation of the artery and the position of the carotid artery in relation to the operator's fingers overlying the larynx are shown in Tables 1 and 2 respectively. The number of attempts to locate the internal jugular vein with both the seeker needle and the introducing needle is recorded in Table 3. In the failed case, the operator was able to enter the vein with the seeker needle but not with the subsequent introducing needle in the three attempts allowed by the study protocol. The supervisor then took over and on his first attempt the vein was entered and successfully cannulated.

Two carotid artery punctures occurred, both with the 21 gauge seeker needle. In each case the vein was successfully located with the seeker needle at the next attempt by aiming the needle in a slightly more lateral direction and the vein was subsequently cannulated without complication. In both these patients the carotid arteries were considered by the operator and the supervisor to be 'just palpable' and to be located directly below the fingers positioned lateral to the larynx.

Table 1. Ease of palpation of the carotid artery with the head facing 45° to the left.

Ease of palpation	Number
Impalpable	5
Just palpable	24
Easily palpable	18
Bounding	3
Total	50

Table 2. Location of carotid artery in relation to larynx.

Carotid artery position	Number
Directly below finger tips with pads of fingers resting on side of larynx	29
One finger-breadth lateral to position above	15
More than one finger breadth lateral	1
Impalpable artery	5
Total	50

Table 3. Number of attempts required to locate the internal jugular vein with the seeker needle and introducing needle.

Number of attempts	Seeker needle	Introducing needle
One	31 (62%)	41 (82%)
Two	14 (28%)	6 (12%)
Three	3 (6%)	2 (4%)
Four	0 (0%)	—
Five	2 (4%)	—
Failure	0 (0%)	1 (2%)
Total	50 (100%)	50 (100%)

The carotid artery was considered to be 'easily palpable' in only 21 (42%) cases, and only 'just palpable' in 24 (48%). It was 'impalpable' in five (10%) of the patients studied.

The position of the head in which the carotid artery was most readily palpable was recorded in 27 of the 50 patients. In three (11%) of the patients it was noted that the artery was easier to palpate with the head facing forward, whilst in the remaining 24 (89%) ease of palpation was not affected by head position. In the five patients with impalpable carotid arteries, the internal jugular vein was located successfully by the seeker needle in a mean of 1.4 attempts (range 1–2) and with the introducing needle in a mean of 1.2 attempts (range 1–2). In comparison, in the 45 patients in whom the artery was palpable there were a mean of 1.6 attempts (range 1–5) with the seeker needle and 1.3 attempts (range 1–3) with the introducing needle respectively. No arterial punctures occurred in patients with impalpable carotid arteries. No problems were encountered with feeding the wire through the 18 gauge needle in any patient.

In two patients problems were noted during the cannulation procedure. In the first there was an error of technique by the operator in that the fingers of the left hand were initially positioned 3 cm too far laterally. This error was only recognised after four attempts to locate the vein had been made with the seeker needle. The fingers were then correctly repositioned and the vein successfully located on the next attempt. In the other patient, although the vein was located at the second attempt with the seeker needle, the operator could not subsequently enter the vein with the introducing needle. After the maximum three attempts dictated by the protocol, the supervisor took over and successfully entered the vein at the first attempt. This was the only failure in the study.

Discussion

Using this new technique for cannulation of the internal jugular vein the success rate was 98%. We believe this method is particularly suitable for teaching anaesthetic staff-in-training. The value of using a small gauge seeker needle is emphasised. Two cases of arterial puncture occurred, both using the 21 gauge seeker needle, and were easily recognised; the vein in each case was subsequently successfully cannulated with the introducing needle without complication. No carotid artery punctures occurred with the 18 gauge introducing needle. Both of the arterial punctures occurred in patients in whom the carotid arteries were just palpable. A technique which relies solely on palpation of the carotid artery may be difficult in those patients in whom the artery is barely palpable or may be impossible if

the artery cannot be palpated at all. It is of interest to note that arterial punctures did not occur in the five patients in whom the carotid artery was impalpable. If there is doubt whether the needle is in an artery rather than the vein it is recommended that the pressure waveform be observed.

Our technique, if closely followed, can be used successfully by inexperienced operators. Twenty-five SHOs and registrars took part in the study (average estimated previous number of internal jugular cannulations was 120, with a range of 4 to 500). Approximately 40% of the operators commented on the value of identifying and marking reference points before 'draping' the patient, thus avoiding the need to move the drapes subsequently to check the location of landmarks with potential loss of sterility. Another comment noted by 20 of the operators was that they would use this technique in future, especially if there was difficulty in palpating the carotid artery.

The main advantage of this method over other commonly used methods of cannulating the internal jugular vein is that it relies on the location of the cricoid and thyroid cartilages and not on visualising potentially obscure landmarks such as the sternomastoid muscle or the internal jugular vein. It is also a mid to high technique so that the risk of creating a pneumothorax is reduced [1].

Jugular venous pulsations have been used as the sole landmark for percutaneous cannulation, but there may be difficulty in identifying the venous pulsation in awake patients, particularly if the sternomastoid muscle is tense [2]. In addition, venous pulsations may not be visible in obese patients and it has been recommended that pulsations in the neck should not be used to locate the venous pulse in pyrexial and tachycardic patients [2]. The para-carotid approach, as recommended by Oda [3] or Mostert [4], would not be applicable in the 10% of patients with impalpable carotid arteries. Our success rate was similar to that of other techniques of cannulation [1]. Previous studies have demonstrated that inexperienced operators fail to cannulate the vein more often than experienced ones, and that inexperienced operators caused less complications in anaesthetised, ventilated patients than in awake patients [5].

In conclusion, the results of our study reflect the two most important advantages of the described technique: its high success rate and the ease with which it can be performed, using readily identifiable landmarks. Relatively inexperienced operators are particularly likely to find the technique of benefit.

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The caudal boundary of the thoracic paravertebral space

A study in human cadavers

P. A. LÖNNQVIST AND U. HILDINGSSON

Summary

The caudal limit of the thoracic paravertebral space was examined in 13 human cadavers. The origin of the psoas major muscle was found to completely seal off the paravertebral space below the level of the 12th thoracic vertebra. The possibility of caudal spread of a thoracic paravertebral block below this level through the paravertebral space would appear unlikely.

Key words

Anaesthesia, regional; paravertebral block.

Anatomy; paravertebral space.

Despite encouraging reports on the use of thoracic paravertebral block in thoracic, renal and biliary surgery [1–3] the anatomy of the paravertebral space is poorly described in standard anatomical textbooks. Studies investigating paravertebral and intercostal blockade have only addressed the segmental limits of the space (Fig. 1) or examined the potential for free communication between adjacent spaces [1, 4–8]. The cranial and caudal limits of the thoracic paravertebral space have not been defined. We have found consistently, both clinically and radiologically, that thoracic paravertebral blockade does not extend further caudally than the 12th thoracic vertebra (T_{12}) and we propose that some anatomical structure restricts the caudal spread of local anaesthetic solution. In order to test this hypothesis we decided to perform a cadaver study.

Material and methods

The study was carried out in 13 eviscerated human cadavers (10 adults, one child, one infant and one neonate). The caudal boundaries of the thoracic paravertebral space were examined by probing of the space and dye injection and dissection.

Probing

The paravertebral space was approached internally from the thoracic cavity and was located at T_{10} using a standard intravenous cannula (adults, Viggo 1.6 mm OD, UK; children, Viggo 1.0 mm OD, UK). A straight, soft tipped

central venous guide wire (adults, Secalon Seldy, Viggo, UK; children, Certofix Päd, B. Braun, Germany) was gently advanced in the caudal direction until it showed any signs of curling or kinking and this position was noted. The above described sequence was repeated three times in each cadaver.

Dye injection and dissection

Following removal of the guide wire an intravenous catheter was inserted and advanced caudally so that its tip was judged to be just above T_{12} . An injection of methylene-blue (adults, 10 ml, child/infant, 5 ml, neonate, 2.5 ml) was made and the spread of the dye was observed (Fig. 2). The dye was allowed to distribute for 10 min. Psoas major was then transected at L_2 and reflected in a cranial direction by dissection from the vertebral bodies and their transverse processes. The substance of the muscle and the angle between the vertebral bodies and transverse processes were continuously inspected for dye.

Results

Probing

The guide wire could not be advanced beyond T_{11} on two occasions and no further than T_{12} on 32 occasions. During four attempts the probe entered the intercostal space below the 12th rib. The guide wire advanced below T_{12} on one occasion and in this case it was seen to dissect on top of the psoas muscle.

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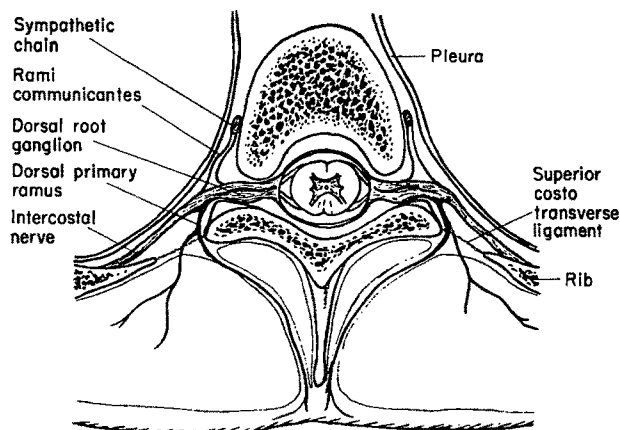


Fig. 1. Transverse section of the thoracic paravertebral space [1] with kind permission from the publisher.

Dye injection

In the great majority of cases the dye filled the paravertebral space at the level of T_{11} and T_{12} , with extensions out into the 11th and 12th intercostal spaces. In three subjects the dye spread to T_{10} but spread across the midline only occurred in one case. On one occasion a small amount of dye was found below T_{12} (different subject than above). In this instance the dye tracked caudally in a fascial plane above psoas major and was subsequently found to have



Fig. 2. Local spread of dye injected in the paravertebral space of a neonate. p = origin of psoas muscle, d = diaphragmatic muscle, c11/c12 = 11th and 12th rib.

penetrated into the abdominal cavity through a tear caused by the eversion.

Dissection

On no occasion could dye or discoloration be seen in the substance of the psoas muscle or in the angle between the vertebral bodies and their transverse processes.

Discussion

In a previous report we stated that thoracic paravertebral block offered an advantage over epidural blockade by preserving the integrity of the lumbar and sacral nerve roots. Thus, the patient is spared the discomfort of numb, partially paralysed legs and urinary retention [3]. The present study supports our suspicion of an anatomical limitation of the paravertebral space at the level of the 12th thoracic vertebra. The origin of psoas major was found to completely seal the paravertebral space at the lumbar level. The two instances where the probe or dye advanced below T_{12} may be artifactual due to over-vigorous probing/injection. The bulk of the muscle will retard diffusion of local anesthetic solution caudally.

Apart from the possibility of longitudinal distribution within the paravertebral space there is evidence that dye or local anaesthetic solution may spread into the extradural space through the intervertebral foramina and contribute significantly to the extension and effect of the blockade [2, 4]. A thoracic paravertebral block might conceivably involve the upper lumbar nerve roots as a result of this type of spread, although it has not, so far, been reported [5, 9].

Based on the findings in this study we conclude that the origin of psoas major defines the caudal limit of the thoracic paravertebral space. Extension of a thoracic paravertebral block caudally through the paravertebral space would seem unlikely caudal to the twelfth thoracic vertebra.

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CASE REPORT

Phantom limb pain during labour

G. P. DUREJA AND SANDHYA

Summary

We report the occurrence of severe phantom limb pain during labour. The patient, a 27-year-old, had had an above knee amputation performed 6 years earlier following a road traffic accident but had no previous history of phantom limb phenomena. However, during early labour, she complained of a severe phantom limb pain in her amputated leg; a continuous epidural block relieved her of the sensation and pain. The sensation did not return following delivery.

Key words

*Pain; phantom limb.
Anaesthetic techniques, regional; epidural.*

Phantom limb phenomena occur in the majority of patients with any kind of deafferentation such as surgical and traumatic amputation, peripheral nerve or spinal injury [1]. The occurrence of this phenomenon is usually seen immediately after the injury but may appear months or years later [2]. Reappearance of phantom limb pain has been reported during spinal anaesthesia [3], epidural anaesthesia [4] and in other stressful conditions. There are however, to our knowledge, no reports in the literature of phantom limb pain appearing during labour.

Case history

A 27-year-old nurse was admitted to the delivery suite in active labour with a full-term pregnancy. Her left leg had been amputated above the knee 6 years earlier following a road traffic accident and she had a prosthetic limb. She had had one live birth following an elective lower segment Caesarean section performed prior to the amputation. After the amputation, she achieved limited mobility with the help of a prosthesis and later continued her professional career.

During the present pregnancy, she had an uneventful antenatal period reinforced with thorough psychological preparation. The attending obstetrician had decided to allow her to go into spontaneous labour at term.

After about 4 h of labour, the patient complained of severe numbness and dysaesthesia in the amputated left leg stump followed by a phantom limb sensation. This gradually became painful and unbearable, the pain being

very intense and 'crushing' in quality. Due to the severity of pain, the patient demanded a termination of labour. In order to attempt to ease the pain it was decided to administer a continuous epidural analgesia. An epidural catheter was inserted at the L₃₋₄ vertebral interspace under aseptic conditions and 10 ml of 0.25% bupivacaine was administered. Within 10 min the patient had complete relief from the painful phantom limb as well as from labour pain. The phantom limb sensation reappeared after 2 h as the effect of the local anaesthetic wore off. Subsequently, three top-up doses of 8 ml of 0.25% bupivacaine were administered at regular intervals, the last dose being given with the patient in the sitting position. The fetus was delivered vaginally with the help of a midcavity forceps. Although the epidural catheter was not removed for 48 h following delivery, the patient did not require any top-up dose during this period. A follow-up was carried out 3 weeks after delivery and she did not have any recurrence of the 'phantom limb' sensation during that period.

Discussion

Phantom limb pain is one of the most distressing of all pain phenomena, yet its underlying mechanisms remain a mystery. Most amputees report a phantom limb sensation almost immediately after amputation of a limb. However, it is only about 35% of amputees who report pain in the phantom limb at some time [2]. The pain may be continuous or occasional, and is described as cramping,

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shooting, burning or crushing. Emotional disturbance is reported to be one of the major factors which may trigger the pain in an amputee [1, 5]. Use of a prosthesis and a lower age at amputation appears to be associated with a lower incidence of such pain [5]. Our patient used a prosthesis immediately following the amputation.

The mechanisms underlying phantom limb pain have been the basis of controversy over the years. So far, no single mechanism has been able to explain the phenomenon. Peripheral, sympathetic, psychological and central mechanisms probably all play a role in producing the classical phantom pain [6]. Melzack [6, 7] has proposed a theory to explain the pain based on the gate control hypothesis of pain. It is believed that a portion of the brainstem reticular formation exerts a tonic inhibitory effect on transmission at all levels of the somatic projection system. When a large proportion of sensory fibres is destroyed by amputation of a limb, the tonic inhibition decreases. This results in self-sustained activity at all neural levels that can be triggered repeatedly by the remaining fibres. Pain occurs when the output of the self-sustaining neurone pools, which project to brain areas that subserve pain experience and behaviour, reaches or exceeds a critical level.

The process of labour and the associated pain can result in strong psycho-emotional disturbances which may modulate the activity in the closed, self-sustaining neurone loops resulting in phantom limb pain in an amputee. Sensory input blockade by epidural block would result in cessation of activity in these self-sustaining neurone loops and so would produce pain relief [1]. Our patient had complete relief of 'phantom limb' pain following epidural block with 0.25% bupivacaine and there was a recurrence of pain as soon as the effect of the local anaesthetic wore off. However, the observation that spinal or epidural anaesthesia [3, 4] causes recurrence or appearance of phantom limb pain, even in patients who had previously suffered little or no pain, is difficult to explain. The decrease in somatic input after spinal or epidural block could lower

the level of inhibition of closed neurone loops and increase the probability of self-sustaining activity resulting in pain.

During early labour, direct pressure of the downcoming fetal head on the visceral sensory fibres in the lower uterine segment and cervix could have contributed towards triggering the pain. The nociceptive impulses from the lower uterine segment travel through the pelvic and hypogastric plexus to the lumbar and lower thoracic sympathetic chains and the sensory stimuli from the cervix travel along the pelvic splanchnic nerves to the second to fourth sacral segments and the sacral portion of the sympathetic chain. Cronholm [8] has suggested that sensory inputs of this nature, or even a mild painful stimulus at effective sites in some normal areas of the body, can evoke 'phantom limb' pain in amputees. The fact that this patient's pain was relieved by segmental epidural blockade supports this hypothesis.

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CASE REPORT

Ventricular fibrillation after accidental injection of bupivacaine into the pericardium

B. J. EVEN, R. F. DE JONGH AND S. G. DE HERT

Summary

A postoperative cardiac surgical patient developed ventricular fibrillation immediately after accidental pericardial injection of bupivacaine at room temperature. The possible causes, which include systemic toxicity, local vasoconstriction with myocardial ischaemia, local toxic effect of bupivacaine or local hypothermia, are discussed.

Key words

Toxicity; bupivacaine.

Anaesthetic techniques, regional; interpleural block.

Heart; ventricular fibrillation.

The use of interpleural bupivacaine for analgesia in acute and chronic pain has been reported since 1984. The technique is effective after upper abdominal surgery, but only variably so after thoracotomy [1]. It is thought to work by diffusion of local anaesthetic through the parietal pleura and intercostal muscles, thus blocking the intercostal nerves (dermatomes T₂ to T₁₂). The recommended doses of bupivacaine for interpleural analgesia are 20–30 ml in a 0.5 to 0.75% concentration. For treatment of postoperative pain which is resistant to opioids, we have used bupivacaine 0.125% injected into the pleural drain while it is briefly clamped. We report a patient where bupivacaine was accidentally injected through a pericardial drain, with resultant ventricular fibrillation.

Case history

A 64-year-old man had a history of two myocardial infarctions. Left ventricular function was good but coronary angiography showed triple vessel disease. Uneventful complete surgical revascularisation was performed and a left pleural and a pericardial drain were left *in situ*. On the first postoperative day the patient was haemodynamically stable and his pain was treated with piritramide 15 mg intramuscularly. However, on the second day the pain was resistant to this treatment and plain bupivacaine 0.125% 20 ml (25 mg) was injected into the clamped left pleural

drain. After 15 min the pain had not diminished, therefore it was decided to inject an additional bolus of bupivacaine 0.125% 10 ml (12.5 mg) interpleurally. However this bolus was inadvertently injected into the pericardial drain, at which point the patient developed ventricular fibrillation. A single DC shock was sufficient to restore sinus rhythm and stable haemodynamics and there were no further cardiac or neurological complications.

Discussion

Several physiological mechanisms can explain the occurrence of ventricular fibrillation after injection of bupivacaine at room temperature into the pericardial sac.

First of all, a local toxic reaction of bupivacaine on epicardial fibres, or perhaps rapid absorption into the coronary arteries, could be responsible for life-threatening arrhythmias. The electrophysiological effect of bupivacaine on myocardial cells results from an interaction with the fast sodium channels in the cardiac membrane [2]. This phenomenon decreases the maximum rate of depolarisation, the amplitude and duration of the action potential and shortens the effective refractory period. This may result in unidirectional conduction delay, ventricular arrhythmias and fibrillation.

Secondly, rapid systemic absorption of bupivacaine, superimposed on that absorbed following the previous

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interpleural bupivacaine injection, could have induced toxicity. Severe cardiovascular side effects have been described at serum levels greater than 3 to 6 $\mu\text{g}\cdot\text{ml}^{-1}$ [3]. Since no toxic symptoms have been found with 20 ml 0.75% (150 mg) interpleural bupivacaine which resulted in serum levels of less than 3 $\mu\text{g}\cdot\text{ml}^{-1}$ [4], it seems unlikely that the dose given to our patient (37.5 mg) could have induced systemic toxicity. However, it should be remembered that after cardiac surgery, absorption of interpericardial and interpleural local anaesthetics without adrenaline may be increased because of hyperaemia and inflammation of damaged tissues [1]. When bupivacaine is instilled into the pericardial sac it is not bound to serum protein, therefore rapid uptake is possible. These factors may be responsible for serum levels of free bupivacaine which are higher than usual. However, toxic levels of bupivacaine that induce cardiovascular side effects are three to seven times greater than those that produce central nervous excitatory symptoms [2, 5]. Hence, although we did not determine serum levels of bupivacaine in our patient it seems unlikely that systemic cardiac toxicity would have occurred without neurological symptoms.

Local anaesthetic agents exert a biphasic action on the smooth muscle of peripheral blood vessels. Low dose bupivacaine has been shown to induce vasoconstriction of rat muscle arterioles, whereas a bupivacaine concentration of 1 $\text{mg}\cdot\text{ml}^{-1}$ induced vasodilation [6]. Moreover, hypothermia augments saphenous vein reactivity to noradrenaline by an increase in the responsiveness of α -2 adrenoreceptors [7]. Agonists, such as adrenaline and noradrenaline, are present in high concentrations during pain or after cardiac surgery. It is possible that the combination of bupivacaine, local hypothermia (since the fluid was at room temperature) and circulating catecholamines, could have been responsible for vasoconstriction of venous bypasses, the mammary artery or coronary arteries. This might then have provoked myocardial ischaemia that induced ventricular fibrillation.

Local hypothermia of myocardial cells might also be a cause of malignant arrhythmias. A decrease in temperature of 5 degrees or more causes impairment of electrical activity in ventricular fibres. Lowering the temperature reduces the cellular oxidative processes that provide energy for the ion pumps [8] and this reduction of ion pump activity lowers the sodium gradient between the intracellular and extracellular compartment, thus diminishing the resting membrane potential [9]. This can be detected on the electrocardiograph by the appearance of J-waves and a progressive widening of the QRS complex, with concomitant QT interval prolongation.

A combination of hypothermia and drugs such as bupivacaine can widen the QRS more than might be expected if the bradycardia is associated with hypothermia alone. Both hypothermia and bupivacaine may induce unidirectional blockade of the ventricular conducting pathways, resulting in re-entrant arrhythmias. In dogs, a potentiation of the effects of both these factors on the depolarisation time and effective refractory period has been demonstrated [9].

Although the exact mechanism for the development of ventricular fibrillation in this patient cannot be determined, it seems likely that the combined local effect of bupivacaine and hypothermia on myocardial cells precipitated ventricular fibrillation. However, the involvement of local vasoconstriction cannot be totally excluded.

This case of ventricular fibrillation after accidental pericardial administration of bupivacaine emphasises that, before treating severe pain after cardiac surgery by interpleural local anaesthetics, it is essential to check the location of the drain. Secondly, because the pleural and pericardial spaces are frequently fused following cardiac surgery, one can visualise that interpleural bupivacaine has the potential to produce arrhythmias by a local effect.

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CASE REPORT

Are electrocardiogram changes the first sign of impending peri-operative pneumothorax?

G. BOTZ AND J. G. BROCK-UTNE

Summary

A patient in the right lateral position underwent left nephrectomy, after which he was placed supine for insertion of an arteriovenous fistula. All haemodynamic and respiratory values, including peak inspiratory pressure, were within normal limits and unchanged from baseline measurements. However, following the position change we noted that the amplitude of the electrocardiogram complexes were dramatically reduced. Our differential diagnosis included the possibility of a pneumothorax, which was subsequently confirmed by both physical examination and chest X ray. A chest drain was planned to be inserted at the end of the surgery, but 25 min after the electrocardiogram changes were noted, the patient's vital signs suddenly deteriorated. Emergency treatment for pneumothorax was instituted with good effect. The diagnostic use of the electrocardiogram and the treatment of this intra-operative pneumothorax are discussed.

Key words

Anaesthesia; general.

Complication; pneumothorax.

Heart; electrocardiography.

The diagnosis of intra-operative pneumothorax can occasionally be difficult. We report a case where a decrease in electrocardiogram (ECG) amplitude was the first sign of the event. This change occurred 25 min before a rapid deterioration in the patient's clinical condition necessitated urgent treatment.

Case history

A 55-year-old male patient with progressive renal insufficiency and a left renal mass presented for left nephrectomy and arteriovenous (A–V) shunt placement. His past medical history included well-controlled hypertension, Crohn's disease, progressive renal insufficiency, and a history of cigarette smoking. Medications included atenolol and sulfasalazine.

He underwent an uncomplicated induction of anaesthesia and tracheal intubation. A radial arterial line was placed after induction of anaesthesia and he was positioned in the right lateral decubitus position for surgery. He remained haemodynamically stable during the nephrectomy with a mean arterial pressure (MAP) of 75–85 mmHg, and an oxygen saturation (SpO_2) of 98%.

The peak inspiratory pressure was increased slightly from 14–16 cmH₂O in the supine position to 20–22 cmH₂O when he was in the lateral position. The ECG demonstrated sinus rhythm and was similar to the pre-induction ECG (Fig. 1). Following nephrectomy he was moved to the supine position in preparation for the A–V shunt placement. Re-assessment of airway and monitors showed no changes. Ten minutes later, during the preparation of the left upper arm, the lead II ECG tracing was noted to be dramatically reduced in amplitude (Fig. 2) when compared to the pre-induction tracing (Fig. 1). Re-examination of the

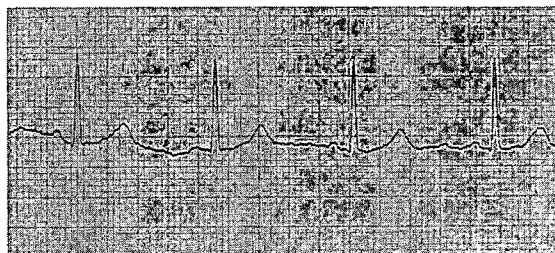


Fig. 1. The ECG before induction of anaesthesia.

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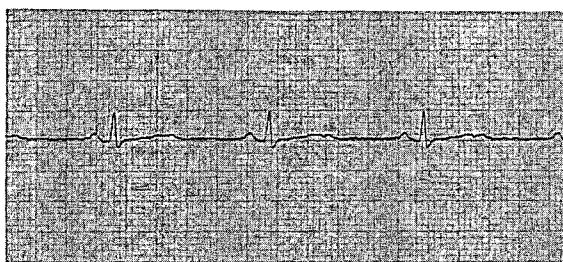


Fig. 2. The ECG after nephrectomy and position change (see text).

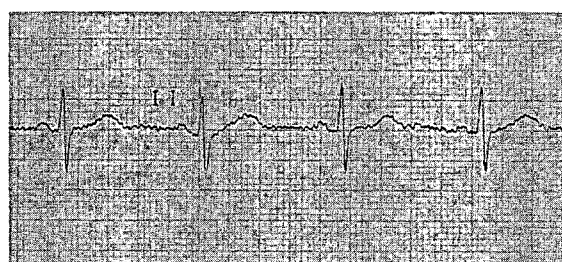


Fig. 4. The ECG after medial placement of electrode.

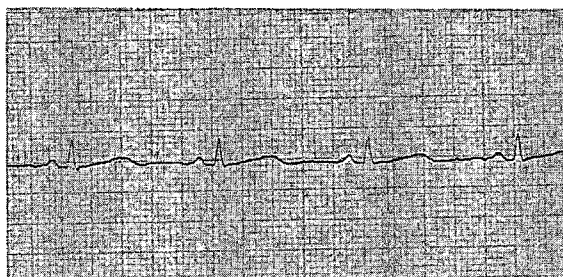


Fig. 3. The ECG after emergency pneumothorax drain insertion.

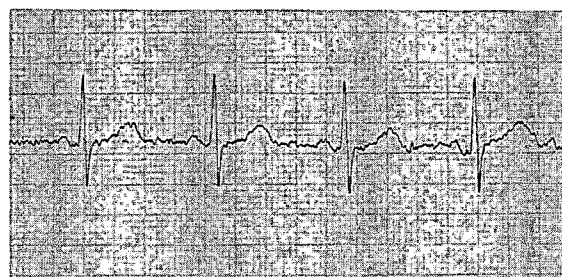


Fig. 5. The ECG after placement of chest drain.

patient revealed decreased breath sounds and hyperresonance to percussion over the left thorax. Anaesthetic gases were changed from 70% nitrous oxide in oxygen to 100% oxygen with isoflurane 0.5%. His haemodynamic measurements remained stable. A portable chest X ray revealed a 1–2 cm pneumothorax on the left side.

Since the patient's condition remained stable, it was decided to place the chest drain at the end of surgery. However, 25 min after the diagnosis of a pneumothorax, without any further change in the amplitude of the ECG, there was a sudden decrease of SpO_2 to $< 95\%$ and the mean arterial pressure decreased to 40 mmHg with a dramatic increase in peak inspiratory pressure from 25 to 58 cmH₂O. A 14-gauge intravenous cannula was inserted rapidly through the left second intercostal space at the midclavicular line resulting in an audible escape of air. The catheter was attached to an emergency underwater seal [1]. Within 2 min the SpO_2 , peak inspiratory and mean arterial pressures returned to the previous values. No change was seen in the ECG amplitude following this manoeuvre (Fig. 3). At the end of surgery, the left chest ECG electrode had to be moved, as it was in the way for the proposed insertion of a conventional chest drain. Therefore a new electrode was placed several centimetres medially, explaining the change in ECG complex morphology (Fig. 4). After the placement of the chest drain, the ECG amplitude increased to near baseline levels (Fig. 5). The patient made an uneventful recovery and was discharged home one week later.

Discussion

ECG changes produced by spontaneous pneumothoraces have been well described as a diagnostic aid in the awake, spontaneously breathing patient [2–4]. However, in anaesthetised patients developing an intra-operative pneumothorax [5–7], this sign has not been alluded to, except in one case report [8]. Anaesthetists therefore have relied on changes in haemodynamic and respiratory values to make a

diagnosis of intra-operative pneumothorax [9]. We believe that our case, together with a previous similar case report [8], may indicate that ECG changes occur early and may be the only sign of an impending intra-operative pneumothorax in anaesthetised patients; the ECG changes manifesting themselves before the pneumothorax becomes life threatening. It would therefore seem prudent, especially following procedures where spontaneous pneumothorax may be a complication, that careful attention be paid to any ECG changes. In our case the dramatic drop in QRS amplitude made us consider the possibility of a pneumothorax. Physical examination and chest X ray confirmed our suspicion. We elected to wait to treat the pneumothorax since all other measurements remained normal. In our case cardiovascular and respiratory values suddenly deteriorated necessitating urgent treatment. Hence, we believe that urgent physical examination and/or chest X ray should be performed despite surgical constraints, when ECG changes occur pointing to a possible pneumothorax. However, it is important to realise that the X ray may also be misleading as it may have to be taken with the patient supine and fail to demonstrate air in the chest.

The case report by Ruo and Rupani [8] mentions that a decrease in the QRS amplitude was the first sign noted of their pneumothorax. Similar to our case, their patient's other cardiovascular and respiratory parameters remained unchanged for an unspecified period of time (most likely more than 1 h). Since they considered the possibility of a cardiac ischaemic episode surgery was allowed to continue, fortunately without untoward effects. At the end of surgery (time unknown) and while the patient was being ventilated, the chest was noted to move asymmetrically during inspiration. A chest X ray confirmed the diagnosis of pneumothorax. After the chest drain was placed the ECG reverted to the pre-operative size, as did the ECG in our patient. At no time did their patient develop other haemodynamic or respiratory changes consistent with a life-threatening pneumothorax. We believe that the ECG changes seen in their patient are similar to

our case, implying that the first indication of intra-operative pneumothorax may be a decrease in amplitude of the ECG. The case described by Ruo and Rupani [8] had a left pneumothorax, as did ours. It is possible that right pneumothoraces may not give such a dramatic reduction in ECG amplification as left ones. The ECG changes with right pneumothoraces may occur later and/or give different ECG patterns.

In conclusion, should nonspecific ECG changes, especially a decrease in amplitude, be seen intra-operatively it may be prudent to insist that a detailed chest examination and possibly a chest X ray be performed.

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CASE REPORT

Anaesthesia for thoracoscopic pleurectomy and ligation of bullae

F. A. MILLAR, G. L. HUTCHISON AND R. A. B. WOOD

Summary

A patient is described who, despite severe pre-operative respiratory disability, had her persistent pneumothorax successfully managed by thoracoscopic pleurectomy. The technique causes considerably less pain and interference with respiratory function postoperatively than does conventional thoracotomy. Potential anaesthetic problems arise because of the necessity of insufflating carbon dioxide at pressures of up to 1 kPa to maintain a pneumothorax during surgery.

Key words

*Anaesthetics, intravenous; propofol.
Equipment; computers, infusion systems.
Complications; pneumothorax.
Surgery; thoracoscopic pleurectomy.*

The videothoracoscopic technique for pleurectomy and ligation of bullae in patients suffering from recurrent pneumothorax has been described by Nathanson *et al.* [1]. Surgery is carried out through four cannulae. Two 11 mm diameter cannulae are used for the insertion of a telescope and/or instruments, and two cannulae 5.5 mm in diameter are used for instruments only. To allow surgery to be performed, a pneumothorax is maintained by the continuous pressure-controlled insufflation of carbon dioxide.

The technique has several advantages over thoracotomy. Postoperative pain is minimised, and reduced surgical handling of the lung may lead to more rapid re-expansion. Both these features result in minimal postoperative respiratory deficit and early mobilisation. Patients are discharged from hospital and return to normal activity sooner [1].

We wish to illustrate the advantages of this procedure by reporting a patient whose persistent pneumothorax was managed successfully despite severe pre-existing respiratory impairment.

Case history

A 71-year-old woman with a history of severe chronic obstructive airways disease and, one year previously, bilateral pulmonary tuberculosis, was admitted to hospital as an emergency with an acute onset of dyspnoea. A chest

X ray revealed a partial left pneumothorax, drainage of which relieved her symptoms. Although the lung re-expanded, an air leak through the drain persisted for 3 weeks, at which stage surgical intervention was considered.

At this time, arterial oxygen tension (P_{aO_2}) while breathing air was 6.0 kPa, with an arterial carbon dioxide tension (P_{aCO_2}) of 6.1 kPa. The patient was of short stature, with a height of 150 cm, a weight of 37 kg, and a marked kyphosis. Her forced expiratory volume in one second (FEV_1) was 0.75 l, with a forced vital capacity (FVC) of 1.4 l ($FEV_1/FVC = 0.53$). Total lung capacity was 3.2 l. Exercise tolerance prior to admission had been limited (by breathlessness) to short distances on the flat, but was sufficient to allow her to get around the house. She was being treated with imipramine for chronic depression and ranitidine for previous peptic ulceration. The electrocardiogram (ECG) showed P pulmonale but was otherwise normal. The haemoglobin concentration was 15 g.dl^{-1} , and other routine haematological and biochemical tests were within the normal ranges.

The surgeon considered an open thoracotomy to be inappropriate in view of her respiratory deficit, but her case was suitable for the recently developed thoracoscopic approach to repair the air leak and perform pleurectomy.

Premedication was with temazepam 10 mg. In the anaesthetic room, ECG, pulse oximetry and noninvasive blood

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pressure monitoring were instituted. An intravenous infusion was established through a 14 G cannula. Pre-oxygenation with 100% oxygen was applied for 3 min, by which time the pulse oximeter recorded 97% oxygen saturation.

Induction of anaesthesia was carried out using a computer-controlled propofol infusion system, designed by White and Kenny [2, 3]. This system consists of a Psion Organiser computer driving an Ohmeda 9000 pump. The computer uses a three-compartment model of propofol pharmacokinetics (based on the patient's age and weight) to calculate the propofol dose required to achieve a desired blood propofol concentration. Frequent recalculations then led to a constant tailoring of the propofol infusion rate to maintain the desired blood levels. The use of this system has previously been described for the induction of anaesthesia in the presence of an undrained pneumothorax [4] and a bronchopleural fistula [5]. In this patient, an initial subanaesthetic target blood propofol concentration of $1 \mu\text{g}\cdot\text{ml}^{-1}$ was set. Target levels were then increased slowly in $0.5 \mu\text{g}\cdot\text{ml}^{-1}$ increments to $3.5 \mu\text{g}\cdot\text{ml}^{-1}$, at which level the patient lost consciousness. She breathed spontaneously throughout induction, and the oxygen saturation remained 97%. Gentle, positive, pressure-assisted ventilation by hand produced swinging of the underwater sealed drain, but no air leakage. The lungs ventilated easily and symmetrically. Suxamethonium 30 mg was administered to facilitate intubation. It was found to be impossible to adequately position a double lumen tube. A 7.5 mm tracheal tube was passed, and it was found that this could easily be advanced into the right main bronchus to isolate the left lung if required. Since the lungs were still easily ventilated, it was decided to proceed without further attempts to pass a double lumen tube.

Anaesthesia was maintained with 0.75% isoflurane and 50% oxygen in nitrous oxide, and intermittent bolus doses of papaveretum. Muscle relaxation was by atracurium infusion. Radial artery and left internal jugular cannulae were inserted for invasive pressure monitoring, and end-tidal carbon dioxide concentrations were measured. When the patient had been positioned on her right side, the tracheal tube was checked to ensure that right bronchial intubation could still be performed if necessary.

Carbon dioxide from an electronic Storz insufflator, set at 6 mmHg (0.8 kPa) pressure, was attached to the chest drain to push the lung away from the chest wall while ventilation was maintained. Cannulae were inserted as described by Nathanson *et al.* [1] and, with a 30° vision telescope 10 mm wide, the lung was fully inspected. Severe fibrous change was seen at the apex of the lung, but no leak or bulla was detected. Parietal stripping was then performed from the apex of the pleural cavity to 8 cm above the insertion of the diaphragm anteriorly and posterolaterally. A new chest drain and an interpleural catheter for postoperative pain relief were inserted under telescopic vision, and the lung was then observed to re-inflate easily and fully. Bupivacaine 0.5% 10 ml were instilled through the interpleural catheter once the patient had been returned to the supine position.

In view of her poor respiratory reserve, the patient was transferred to the intensive care unit for postoperative monitoring. She was weaned from ventilation without difficulty 6 h later, spent an uneventful night, and returned to the surgical ward the next morning. Arterial blood gases at

that time were Pao_2 13.2 kPa and Paco_2 5.7 kPa on 35% oxygen by face mask. Interpleural local analgesia was continued for 48 h after surgery, supplemented by three doses of papaveretum 8 mg intramuscularly.

She remained weak, with a poor appetite and her serum albumin concentration was $30 \text{ g}\cdot\text{l}^{-1}$. Mobilisation was slow and she required prolonged physiotherapy to overcome Achilles tendon contractures, which were a consequence of her short stature and pre-operative immobility: when she sat on a chair, her feet did not always reach the floor. She was discharged home on her 30th postoperative day.

At the time of her first surgical outpatient review, she had returned to her limited pre-operative level of activity.

Discussion

Although inhalational induction of anaesthesia has been the standard approach to patients with actual or potential air leaks from the lung, a computer-controlled infusion of propofol has been used successfully [4, 5]. The ability of this system to increase blood propofol concentrations in small increments, with minimal overshoot, allows the patient to be anaesthetised without episodes of apnoea or exposure to nitrous oxide.

We were unable to position a double lumen tube satisfactorily in the patient described, perhaps because of her short stature and kyphosis, but surgery was easily carried out, despite the use of a simple tracheal tube. Full deflation of the operated lung is neither necessary nor desirable, since it makes identification of bullae more difficult. A pneumothorax, sufficient to allow thoracoscopy and surgery, is maintained by the introduction of carbon dioxide through an insufflator, with a cut-off pressure set to 6–8 mmHg (0.8–1 kPa) or less.

It is our normal practice to insert a double lumen tube for such cases. This allows full control of any air leak, and facilitates partial deflation of the operated lung, minimising the insufflation pressure needed to obtain a pneumothorax of the required size. A gate clamp may be used to partially occlude the limb of the double lumen catheter mount connected to the operated lung. This results in reduced ventilatory movements on the operated side during surgery, while the presence of an increased resistance to expiration holds the lung partially inflated. Preferential ventilation of the opposite lung may also serve to reduce the mediastinal shift caused by the presence of pressurised gas in the operated hemithorax.

This patient made a prompt respiratory recovery after surgery, despite severe pre-existing lung disease, illustrating the advantage to the patient of thoracoscopic rather than open surgery. We conclude that the thoracoscopic approach to pleurectomy and ligation of bullae has significant advantages for the patient, in terms of reduced pain and preservation of respiratory function. However, the presence of pressurised gas in the operated hemithorax has the potential to induce major physiological disturbances, so that careful monitoring and meticulous control of gas pressures are required throughout the procedure.

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CASE REPORT

Transoesophageal echocardiography during removal of a caval filter in a patient at high risk of massive pulmonary thromboembolism

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Summary

A new type of vena caval filter was to be removed from a woman who had a high risk of massive pulmonary thromboembolism because of extensive thrombus in the iliac vein incorporating the filter. The removal was performed under general anaesthesia in the cardiac operating theatre using transoesophageal echocardiography to monitor the right heart and the pulmonary artery during the critical phase of removal. The manoeuvre succeeded with only insignificant embolisation occurring, and that was identified by transoesophageal echocardiography.

Key words

*Complications; pulmonary thromboembolism.
Monitoring; transoesophageal echocardiography.
Equipment; removable caval filter.*

Extensive thrombosis involving the iliac vessels poses a significant risk of embolisation to the lungs and may be treated by the insertion of a caval filter. However, if the filter becomes involved in the thrombus, its removal may be hazardous, with the risk of material becoming dislodged; should this occur, rapid surgical treatment is indicated [1–4]. Transoesophageal echocardiography (TEE) can accurately detect emboli passing through the right heart and into the main branches of the pulmonary artery [5–8]. We report the management of a patient, where a new vena caval filter with massive adherent thrombus was removed under general anaesthesia and TEE monitoring.

Case history

A 53-year-old female patient with a history of recurrent episodes of thrombophlebitis was admitted to hospital suffering from endometrial carcinoma and deep vein thromboses in both lower limbs. There was a suspected heterozygous factor XII deficiency; antithrombin III and protein C inhibitor activity were within normal range. To reduce the risk of thromboembolism during hysterectomy, a new removable vena caval filter (Filcard International) was inserted into the inferior vena cava via a right antecubital vein and positioned below the renal veins. Six days after an uneventful hysterectomy, the insertion site for the

filter became inflamed and the patient was transferred to the radiology department for removal of the caval filter. Venography of the inferior vena cava showed complete occlusion of the right iliac vein with thrombus up to, and incorporating, the caval filter (Fig. 1). Local fibrinolysis was ineffective and systemic fibrinolysis was not indicated. In this situation there was a high risk of massive thromboembolism during the removal of the filter. A treatment plan was formulated whereby the filter was to be removed with the patient under general anaesthesia in the cardiac operating theatre with a surgical team and cardio-pulmonary bypass capability available. Removal was to be monitored by transoesophageal echocardiography. After induction of anaesthesia with etomidate and fentanyl and relaxation with atracurium, the patient's trachea was intubated and her lungs ventilated with an oxygen–air mixture. Monitoring consisted of two-lead electrocardiography (II, V₅), central venous pressure, pulse oximetry, capnography and nasopharyngeal temperature. For visualisation of possible thromboembolism, a transoesophageal echocardiographic transducer (5 Mhz) was placed in the oesophagus, connected to a Hewlett-Packard ultrasonic system, which had two-dimensional and Doppler colour flow imaging capabilities. The probe was positioned for monitoring the four-chamber view with special preference to the right heart.

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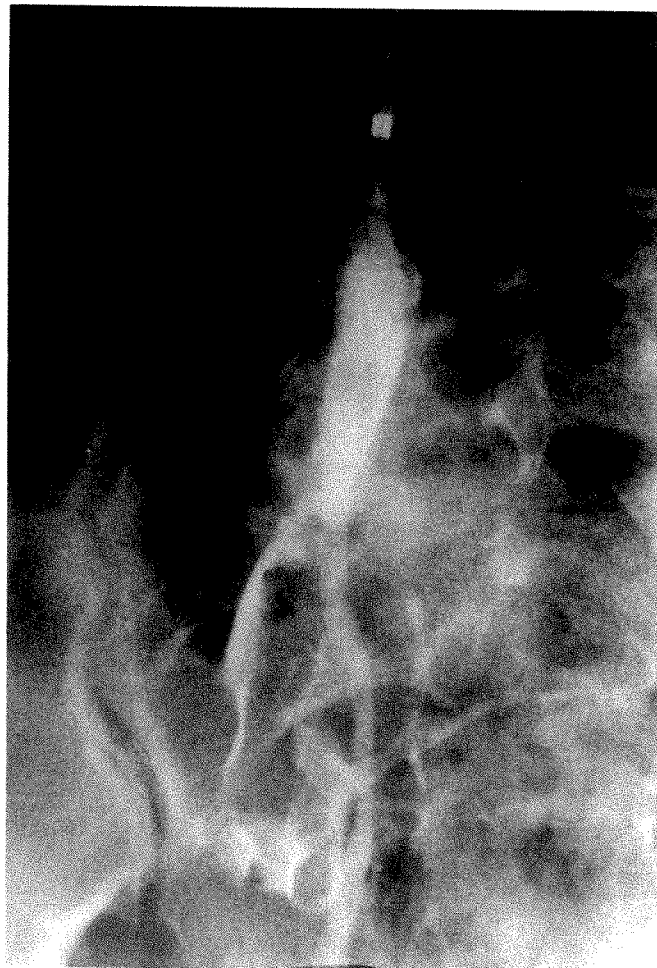


Fig. 1. Angiograph showing massive thrombotic material at and below the caval filter.

The removal of the filter and its passage through the right atrium was uneventful. There was no massive thromboembolism, but 2 min after removal a single 3×5 mm

thrombus entered the right heart, moved around like a 'ping-pong ball', and disappeared (Fig. 2); there were no haemodynamic or respiratory changes. Anaesthesia was

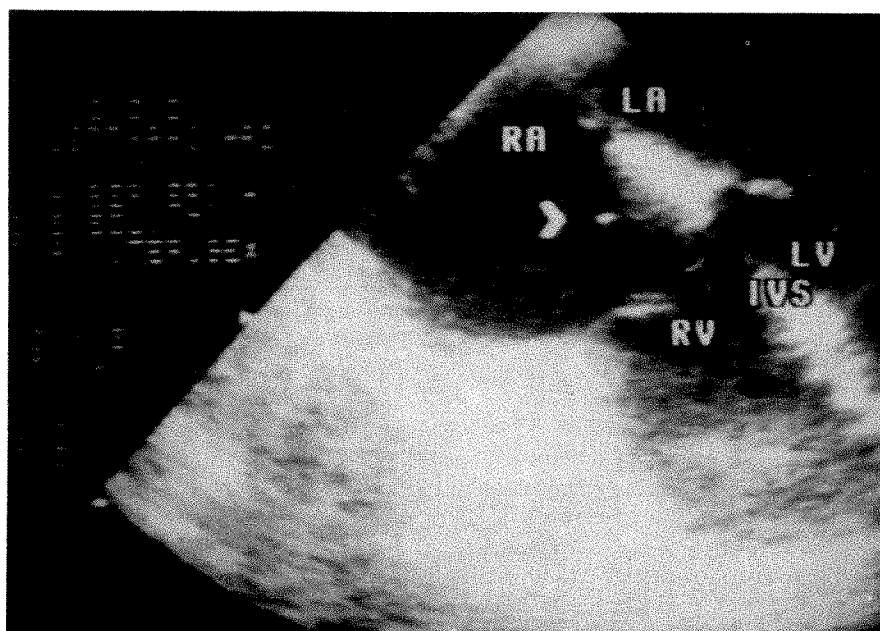


Fig. 2. Transoesophageal four-chamber view with a thrombus in the right atrium immediately before passing through the tricuspid valve. RA, right atrium; LA, left atrium; RV, right ventricle; LV, left ventricle; IVS, interventricular septum.

discontinued after 15 min, and the patient's trachea was extubated 10 min later. She was transferred to the intensive care unit and discharged to the ward after 24 h.

Discussion

Permanent inferior vena caval filters have been implanted for many years [9]. A new, removable vena caval filter has been designed for severe thromboembolic disease allowing immediate local fibrinolysis and protection from pulmonary embolism. There have been no reports about the management of this removable vena caval filter in cases of massive thrombus incorporating the filter and associated with unsuccessful local fibrinolysis. Rapid surgical intervention is indicated in the event of massive pulmonary embolism if local or systemic fibrinolysis is ineffective or contraindicated. Indications for surgical intervention include occlusion of more than 50% of the pulmonary vascular cross-sectional area and occlusion of the right or left pulmonary artery [1-4]. If removal of a vena caval filter (with adherent thrombus) is considered to entail a high risk of massive pulmonary embolism, it may be appropriate to remove the filter with the patient under general anaesthesia and a surgical team available. In this situation TEE can monitor the removal of the filter, detect thrombus in the right heart, and diagnose large emboli in the main branches of the pulmonary artery. Recent reports have demonstrated that TEE is an accurate and safe diagnostic tool for the visualisation of thromboembolism [5-8]. Most reports have referred to patients undergoing hip replacement. Klein *et al.* described visualisation of acute pulmonary emboli by TEE during cardiac surgery [6]. McGrath *et al.* reported two cases of massive pulmonary embolism following tourniquet deflation, one of which was documented by TEE [7]. Thrombi or tumour material in the superior and inferior vena cava can also be visualised by TEE [10, 11].

In conclusion, this case report confirms the value of TEE for monitoring patients at high risk of pulmonary embolism. General anaesthesia facilitates monitoring and

increases safety by permitting immediate surgical intervention. However, the indications for, and risks of, removable vena caval filters remain to be fully debated.

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The use of a checklist for anaesthetic machines

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Summary

The use of the Association of Anaesthetists of Great Britain and Ireland checklist for anaesthetic machines, based on an oxygen analyser, was surveyed over a 5-week period in a teaching hospital. Fifty-five completed checklists were analysed; no problems developed during anaesthesia which were missed by the checklist. The mean time taken to complete the checklist for one machine was 8.9 min with a range of 5 to 19 min; for two consecutive machines it was 18.25 min with a range of 10 to 30 min. The most frequent faults detected were the poor reliability of some oxygen analysers, absent ventilator disconnection alarms, and absent oxygen supply failure alarms on some older machines. Faults were found in 60% of the machines checked; 18% of these were deemed to be serious.

Key words

Equipment; anaesthetic machine.
Safety; checklist.

Recently published guidelines have reiterated the desirability of a formal check of anaesthetic equipment before use [1]. The anaesthetists in our hospital always perform some kind of pre-use check of the anaesthetic machine, but in the past it was never formally recorded. The Association of Anaesthetists of Great Britain and Ireland (AAGBI) proposed a checklist which 'takes only a few minutes to perform' and recommend that 'a record of the checks performed should be kept' [1]. In order to assess the time taken to perform the check, the frequency and types of fault detected by this checklist, and any missed faults, we carried out a survey in our hospital.

Methods

A printed checklist (Appendix) was prepared based on the AAGBI recommendations [1]. A suite of six operating theatres (including one 'emergency' operating theatre) was chosen for the study. Each location had one anaesthetic machine in the operating theatre and one in the anaesthetic room (a total of 12 machines). All members were asked to complete, during a specified 5-week period, a checklist for each machine before each operating theatre session. Starting time, finishing time, faults noted during checking and faults noted subsequently during anaesthesia were requested.

Results

A total of 14 anaesthetists returned 57 checklists. One checklist was incomplete and another was completed

outside the study period; these were discarded, leaving 55 forms for analysis. The faults discovered are listed in Table 1. A serious fault was defined as a defect which alone could have led directly to patient harm. Other faults were defined as defects that would not of themselves cause harm but if combined with abnormal circumstances would become dangerous (e.g. a lack of a disconnection alarm is only dangerous if there is an otherwise undetected disconnection). There were a total of 50 faults found, nine (18%) of which were considered serious. On 13 (24%) occasions the oxygen analyser was found to be missing from the machine; one further oxygen analyser was unable to be calibrated and was rejected. On 14 (25%) occasions the ventilator disconnection alarm was missing, and on nine (16%) occasions an oxygen failure alarm was absent. The number of faults and the location of the machines is shown in Table 2.

The time taken for each test is shown in Table 3. The average time to check one machine was 8.9 min with a range of 5 to 19 min. Problems relating to oxygen analysers are shown in Table 4. There were 12 occasions where the same person checked two machines in sequence. The time taken for this is shown in Table 5. The average time to check two consecutive machines was 18.25 min with a range of 10 to 30 min.

Discussion

Whether the recommended checklist of the AAGBI is any more effective than the individual anaesthetists own pre-use

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Table 1. Problems detected during testing.

(1) No ventilator disconnect alarm	14
(2) No oxygen analyser present on machine	13
(3) No oxygen failure warning alarm on machine	9
(4) No pressure relief valve on machine	2
(5) Oxygen pressure gauge not zeroed	2
(6) Leaking valve on manual circuit of ventilator	2*
(7) Cylinders not properly secured	2*
(8) Vaporizers not seated properly	1*
(9) Oxygen analyser not calibrating properly	1*
(10) Oxygen monitor incorrectly fitted in circuit	1*
(11) Bag missing from breathing system	1*
(12) Scavenging not connected to breathing system	1
(13) Suction tubing missing	1*
Total	50

*Potentially disastrous.

Table 2. Number of faults (serious faults) detected for each machine checked.

Site of machine	No of faults detected				Total checks
	No faults	1 fault	2 faults	3 faults	
Anaesthetic room	4	14 (3)	8 (1)	3	29
Operating theatre	18	6 (4)	1	1	26
Total checks	22	20	9	4	55

check of the machine is uncertain. Craig and Wilson studied critical incidents occurring in a District General Hospital and found that 33% of incidents were associated with an inadequate pre-use check of the anaesthetic machine [2] and it has been suggested that pre-use check of the machine may prevent critical incidents [1,3-5]. Carrying out our checklist did detect faults, some of which could have had disastrous consequences (Table 1). This study highlights what may be the case in most NHS

Table 5. Time taken for checking two machines in sequence (min) (second machine checked faster, six times; second machine checked slower, three times; same time for both machine checks, three times).

Doctor	Time taken to check first machine	Time taken to check second machine
C1	15	8
C3	15	12
C3	9	13
C3	10	11
C3	10	7
C3	6	8
C4	8	6
C5	19	11
S1	6	5
R1	5	5
R1	5	5
H3	10	10

C, Consultant; S, Senior registrar, R, Registrar; H, Senior House Officer.

operating theatres: that the anaesthetic machine in the operating theatre is more likely to be new, and thus fault-free (69%), than the anaesthetic room machine (14%). In a survey of 45 hospitals, Kumar *et al.* found that 15.5% of machines did not have a ventilator disconnection alarm and only 64% had a functioning oxygen analyser [6]. The lack of the recommended anaesthetic machine monitoring devices on some older machines highlights the difficulties of trying to keep pace with changes in recommended monitoring standards [7] and the capital outlay necessary to replace older machines which may not have pressure relief valves, oxygen failure warning devices and ventilator disconnection alarms. In the study hospital, older machines without these features tend to be designated to the anaesthetic room.

It is debatable whether this checklist can be described as

Table 3. Time taken to complete checklists (min).

Doctor	Attempt									
	1st	2nd	3rd	4th	5th	6th	7th	8th	9th	10th
C1	15	8								
C2	10	6								
C3	15	12	10	11	9	13	10	7	6	8
C4	9	8	9	9	8	6	7	7		
C5	14	19	11	12						
S1	7	6	5							
S2	8									
S3	13									
S4	8	6	8							
R1	5	5	5	6	5	5				
R2	11									
H1	7	16								
H2	12	6								
H3	10	10	10	10	15	10	5	5	5	7

C, Consultant; S, Senior Registrar; R, Registrar; H, Senior House Officer.

Table 4. Oxygen analyser availability and function.

	Operating theatre	Anaesthetic room	Total	(%)
Present and working	21	12	33	(60)
Present, requiring calibration	5	3	8	(14.5)
Present but not calibrating	0	1	1	(1.8)
Absent, away for repair	2	11	13	(23.6)

'quick' with an average of 18.25 min required to check the usual operating theatre complement of two machines. Compared with the 'single hose and tug' tests [8] which could be performed in under 5 min, it is slow. What is encouraging about the checklist is that although it may be time consuming speed does seem to improve with practice. Checking two machines sequentially is often performed in less than double the checking time for one (Table 5). This may reflect an acute learning process where less frequent reference to the printed checklist is needed for checking the second machine than when checking the first. In addition, some versions of the 'single hose and tug' test did not incorporate an oxygen analyser and would not detect misfilled oxygen cylinders or contaminated pipeline supplies.

The poor reliability of particular oxygen analysers presents another problem with carrying out the recommended checklist. The most reliable method of analysing anaesthetic gases is with a mass-spectrometer, but it is expensive and unsuitable for every operating theatre. The absence of oxygen analysers from some anaesthetic machines was due to the analyser being away for repair (Table 4). On occasions analysers had to be moved with the patient from anaesthetic room to operating theatre.

Not only were faults found in over half of the checklists completed, (60% revealed at least one fault) but serious faults were commonly detected. Table 2 shows that serious faults have a similar likelihood of occurrence in both the anaesthetic room and operating theatre machines. At least 18% of faults had the potential to lead to rapid disaster (Table 1). Of these serious faults only three out of nine could not be readily and safely corrected by the anaesthetist: one oxygen analyser not calibrating properly, and two leaking expiratory valves. These two facts alone should persuade most anaesthetists of the value of 10 to 20 min spent performing a pre-use check. Hopefully, a lack of faults would reflect a high standard of maintenance and a regularly performed checklist is a useful audit for this.

Acknowledgments

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Appendix

CHECKLIST FOR ANAESTHETIC MACHINES

Date: _____ Time started: _____

Theatre: _____

Machine: in theatre/in anaesthetic room (delete as necessary)

Anaesthetist performing test: _____

Assistant (enter name or Nil): _____

This checklist is based on the use of an oxygen analyser and the function of this should be checked first.

(A) Oxygen analyser

Tick if:

checked and OK	checked and sorted
—	—
—	—

1. Function OK?

2. Calibrated?

Attach to common gas outlet (CGO) of machine.

(B) Medical gas supplies

Disconnect all supplies.

Turn vaporizers off.

Turn electricity supply on (if appropriate).

1. Are all cylinders present required?

2. Cylinders secure?

Turn off all cylinders.

Open all flowmeter control valves.

Turn on reserve oxygen cylinder.

3. Contents adequate?

4. Oxygen flowing?

Where second oxygen cylinder is present, turn off reserve cylinder and turn on 'in use' cylinder.

5. Contents adequate?

6. Oxygen flow control fully adjustable?

Set oxygen flow to 5 l.min⁻¹.

7. Oxygen analyser reading 100% (±5%)?

Turn on reserve nitrous oxide cylinder.

8. Contents adequate?

9. Nitrous oxide flowing?

Where second nitrous oxide cylinder is present, turn off reserve cylinder and turn on 'in use' cylinder.

10. Contents adequate?

11. Nitrous oxide flowing?

12. Nitrous oxide flow control fully adjustable?

Set nitrous oxide flow to 5 l.min⁻¹.

Turn off oxygen cylinder.

Press oxygen flush (emergency oxygen) until system empty.

13. Cylinder gauge now zero?

14. Audible oxygen failure alarm working?

15. Oxygen failure protection working? (if fitted)

Connect oxygen pipeline.

16. Oxygen flow restored?

17. Oxygen failure alarm cancelled?

Perform 'tug test'.

18. Oxygen pipeline connection secure?

19. Oxygen pipeline pressure reading 400 kPa?

Turn off nitrous oxide cylinder.

Connect nitrous oxide pipeline.

20. Nitrous oxide flow restored?

Perform 'tug test'.

21. Nitrous oxide pipeline connection secure?	—	—
22. Nitrous oxide pipeline pressure reading 400 kPa?	—	—
Turn off nitrous oxide flowmeter control valve.		
Where fitted:		
Turn on carbon dioxide cylinder.		
23. Contents adequate?	—	—
24. Carbon dioxide flowing?	—	—
25. Carbon dioxide flow control fully adjustable?	—	—
Turn off ALL flowmeter control valves.		
Operate emergency oxygen flush valve.		
26. No significant drop in pipeline supply pressure?	—	—
27. Oxygen analyser reading 100% ($\pm 5\%$)?	—	—
<i>Vaporizers</i>		
28. Vaporizer(s) fitted to machine?	—	—
29. Vaporizer(s) locked to backbar?	—	—
30. Vaporizer control knobs rotate fully?	—	—
Turn off vaporizer(s).		
31. Flow direction through vaporizer(s) correct?	—	—
32. Filling port(s) tightly closed.	—	—
Only perform tests 33–35 where the machine is fitted with a pressure relief valve.		
Set oxygen flow of 6–8 l.min ⁻¹ .		
Turn vaporizer(s) off.		
Temporarily occlude CGO.		
33. No leak detected from vaporizer fitment?	—	—
34. Flowmeter bobbin dips?	—	—
Turn on each vaporizer in turn.		
Temporarily occlude CGO after each open individually.		
35. No leak of liquid from filling port?	—	—
Turn off vaporizer(s).		
Turn off oxygen flowmeter control valve.		

Breathing systems

Inspect breathing system.

36. Configuration (bag, valves etc) correct?	—	—
37. Adjustable pressure limit valve fully mobile?	—	—
Inspect all connections.		
38. Connections tight?	—	—
39. No leaks from pressurised system?	—	—
40. No obstruction in bag/system?	—	—

Ventilator

41. Ventilator operating?	—	—
42. Ventilator controls operating?	—	—
Occlude patient port while operating.		
43. Pressure relief valve operating?	—	—
44. Disconnect alarm present and working?	—	—
45. Alternative means of ventilation available?	—	—

Suction

46. Suction tubing/catheters present?	—	—
47. Adequate negative pressure generated rapidly?	—	—

Time finished: _____

Items of equipment replaced because of fault during checklist: _____

Equipment faults detected during course of the list/procedure: _____

A comparison of the performance of two types of infusion device

S. W. O'KELLY AND J. C. EDWARDS

Summary

The delivery performance of two types of infusion pump, the IVAC 711 series syringe driver and the IVAC 531 series drop-counter, was examined by measuring the output of 16 pumps. We found that despite careful attention to the setting up of all apparatus, the IVAC 711 series syringe drivers had a significant lag phase before output matched the set rate, and that this lag phase was more pronounced at low rates of infusion. This was not the case with the IVAC 531 series which delivered the set rate from the outset. We suggest, that when the timing and rate of an infusion of drug is important from the outset and when these types of pump are being used, that either the syringe driver pump is primed by a brief period of high rate infusion vented by a three-way tap or that a drop-counter pump is used.

Key words

Equipment; infusion pumps.

The practice of giving drugs by infusion has become more common both in the intensive care setting and peroperatively in the operating theatre. Amongst the reasons for giving agents with suitable pharmacokinetic profiles by intravenous infusion is the ability to titrate the infusion rate, and consequently alter blood levels of the drug, in response to perceived changes in the patient's condition. In certain types of anaesthesia, and with certain patients, a rapid response to sudden haemodynamic changes is often necessary. It is therefore of fundamental importance that the anaesthetist is aware of any limitations associated with the apparatus used in providing this response. The performance of some types of syringe pump has been previously examined [1,2]. Stokes *et al.* [1] in their evaluation of the Ohmeda 9000 syringe pump list 10 desirable properties of an infusion device. One of these desirable properties refers to accurate and consistent delivery at flows between 0.1 and 200 ml.h⁻¹.

During anaesthesia for cardiac surgery we frequently use infusions of inotropic agents; these may be required occasionally before cardiopulmonary bypass, and are very often needed after bypass. We have on occasions noticed a delay in drug delivery when using the syringe pump type of infusor and subsequently decided to compare the performance of one example of this type of device with that of a drop-counter pump.

Method

Sixteen infusion pumps were studied. Eight IVAC 711 series syringe driver pumps and eight IVAC 531 series drop-counter pumps. For each pump a new set of accessories was used as directed in the manufacturer's 'Directions for use' manual. We used a standard solution of adrenaline (10 mg in 500 ml of normal saline) as the infused solution for all pumps. Measurement of the pump's output was made by placing the patient end of the extension set within a 10 ml collecting burette marked with 0.1 ml graduations. The end of the extension set was supported in such a way that drops did not make contact with the side of the burette but fell freely to the bottom. A stopwatch was started simultaneously with the pumps and the cumulative delivered output was assessed over intervals of time up to 30 min. The performance of each pump was measured at two rates of infusion—10 ml.h⁻¹ and 20 ml.h⁻¹. The sequence for each pump was randomised and the pumps were switched off and accessories re-assembled between each test period.

Results

Tables 1 and 2 show the time taken by each pump to reach the set infusion rate: either 10 ml.h⁻¹ or 20 ml.h⁻¹. For the

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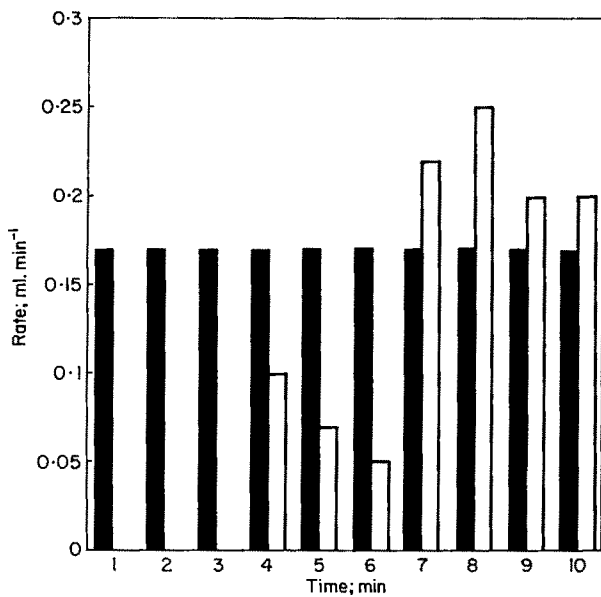


Fig. 1. Delivered rate $\square$ and set rate $\blacksquare$ for series 711 pump when set at 10 ml.h^{-1} .

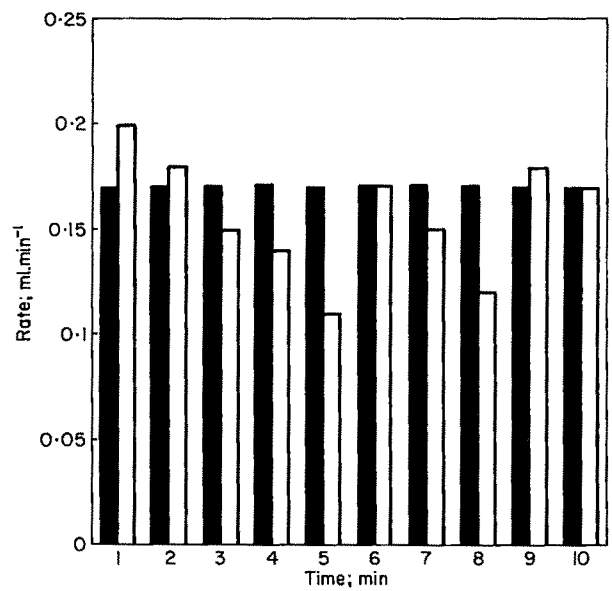


Fig. 3. Delivered rate $\square$ and set rate $\blacksquare$ for series 531 pump when set at 10 ml.h^{-1} .

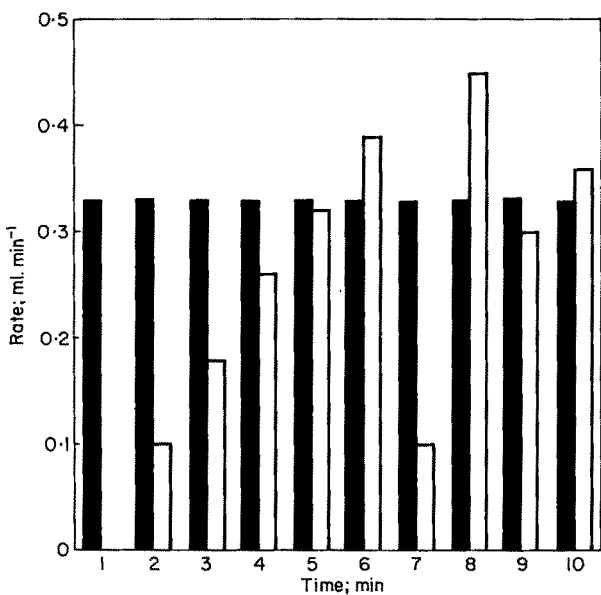


Fig. 2. Delivered rate $\square$ and set rate $\blacksquare$ for series 711 pump when set at 20 ml.h^{-1} .

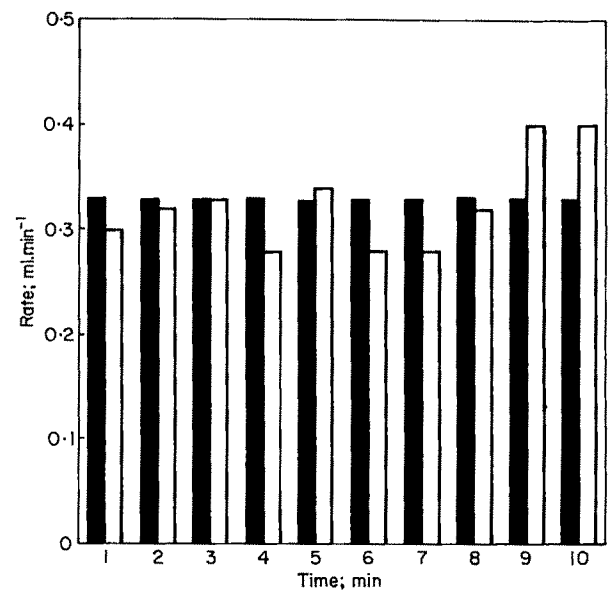


Fig. 4. Delivered rate $\square$ and set rate $\blacksquare$ for series 531 pump when set at 20 ml.h^{-1} .

Table 1. Time taken for each series 531 pump to reach the set rate.

Series 531 Pump No.	Time; min	
	Set at 10 ml.h^{-1}	Set at 20 ml.h^{-1}
1	1	1
2	1	1
3	1	1
4	2	1
5	1	1
6	2	1
7	1	2
8	2	1

Average = 1.4 min Average = 1.1 min
Range (1–2 min) Range (1–2 min)

Table 2. Time taken for each series 711 pump to reach the set rate.

Series 711 Pump No.	Time; min	
	Set at 10 ml.h^{-1}	Set at 20 ml.h^{-1}
1	4	4
2	7	6
3	10	4
4	8	8
5	7	7
6	9	4
7	9	4
8	9	7

Average = 7.9 min Average = 5.5 min
Range (4–10 min) Range (4–8 min)

531 series pumps the average time taken was 1.4 min when set at 10 ml.h^{-1} and 1.1 min when set at 20 ml.h^{-1} . In contrast, when the 711 series pump was set at 10 ml.h^{-1} the average time to reach the infusion rate was 7.9 min and when set at 20 ml.h^{-1} it was 5.5 min. Figures 1 to 4 show these results graphically. Each graph shows the average delivery per min for each type of pump at the two different rates, as well as the delivery expected per min according to the set rate.

Our results show that there is a significant delay before the set rate of infusion is reached when using the syringe driver type of pump; the magnitude of this delay appears to be inversely related to the set rate of infusion. This effect was not seen with the drop-counter type of pump.

Discussion

Accurate drug infusion techniques are becoming more common in many areas of anaesthesia. The ability to respond quickly and appropriately to changes in the patient's condition represents an important part of anaesthetic practice. The accuracy of apparatus used by the anaesthetist in providing this response may often be assumed to be within the manufacturer's claims. While some or all of these claims may be true, certain aspects of performance may not be referred to in the manufacturer's literature. The literature accompanying the IVAC 711 series pump claims a system accuracy of $\pm 5\%$ for volume at rates of 1.0 ml.h^{-1} and above. It also claims an instantaneous response to rate changes. While this may be true for the majority of the time, our results show that at initial set-up there is a period of several minutes before this degree of performance accuracy is achieved. The average delay of 7.9 min to reach the set rate, seen with the 711 series when set at 10 ml.h^{-1} , may represent a significant delay and lead to an unwitting failure by the anaesthetist to respond to an acute change in the patient.

Our study was not designed to determine the cause of the delay we demonstrated. We recognise that with the syringe driver pump, the internal mechanical slack must be overcome before solutions are infused. This may be minimised but not eliminated by the careful placement of the syringe in the driver and application of the syringe clamp. In addition, there may be an initial resistance of the syringe or 'stiction' [1] where the plunger adheres to the inner wall of the syringe barrel. Any small bubbles in the syringe or extension set would also increase the initial compliance and be likely to contribute to a delay.

In conclusion, we would accept that for the majority of occasions that the anaesthetist might use a syringe driver pump, an initial delay would be only of slight clinical importance. Nevertheless we would suggest that there are many occasions when the anaesthetist requires the infused agent to be delivered to the patient with as brief a delay as is possible. This study has shown that on such occasions the anaesthetist's reliance on a syringe driver type of pump to perform as desired may not be well-founded. As a result of our study we now prime the syringe driver by a brief period of high rate infusion vented via a three-way tap before the syringe pump is applied to the patient. It may be that, in addition to the accurate and consistent delivery of solution, another explicit desirable property of an infusion device would be the ability to infuse solution accurately immediately after the device has been started.

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Forum

An evaluation of the behaviour of children undergoing dental extraction under general anaesthesia

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Summary

The cooperation of a random sample of 190 children with an average age of 5 years (range 1–17 years), presenting with a dental emergency requiring tooth extraction under general anaesthesia, was evaluated using the Frankl Behaviour Scale. The results showed that 60% of the children cooperated in the operating theatre. Furthermore, removal of only one or two teeth per patient accounted for 40% of the cases. This survey has identified a group of child dental patients who might benefit from conscious sedation and local anaesthetic techniques.

Key words

*Anaesthesia; paediatric, dental.
Psychological responses.*

General dental practitioners in the UK rely largely on general anaesthesia when treating children, citing poor cooperation and multiple tooth extractions as the main reason. For example, in a 1986 survey of the use of general anaesthesia and sedation in the general dental service in East Anglia [1] half of the respondents used general anaesthesia and/or sedation. Three-quarters of the administrations were for simple extractions, 70% of patients were given a general anaesthetic and nearly 60% of these were administered to children less than 12 years of age.

Over the last 30 years, changes in dental anaesthetic practice have been evolving, partly as a result of the increasingly sophisticated requirements of dental surgeons, particularly in the field of conservative dentistry, and partly as a consequence of the introduction of new anaesthetic agents and techniques. With the improvements in anaesthetic standards, more critical attention has been concentrated on the morbidity and mortality associated with dental anaesthesia, and the examination of their possible causes. These developments have coincided with a decrease in the number of patients requiring outpatient dental anaesthesia in the dental hospitals, so that dental undergraduates received insufficient teaching and practical experience in this field. This problem was compounded by the increasing need to train postgraduates in dental anaesthesia, thus further diluting the available clinical experience [2].

In 1986, Parbrook [3] predicted the death of general anaesthesia in the general and community dental services. Two important factors were the declining incidence of caries and the recommendation from the General Dental Council 'that neither general anaesthesia nor sedation should be employed unless proper equipment for their administration and adequate facilities for resuscitation of

the patient are readily available with both the operator and his staff trained in their use'. He anticipated that the increasing use of alternative techniques, which combined local anaesthesia with sedation using inhaled nitrous oxide or intravenous midazolam, would contribute to its demise.

Wide variation in the demand for a general anaesthetic service exists within the UK. In the two years 1987–8, 3623 general anaesthetics were administered to children in London compared with almost 10 000 in Glasgow [4, 5].

In a recent report of an Expert Working Party on General Anaesthesia, Sedation and Resuscitation in Dentistry [6] it was recommended that general anaesthesia should be avoided whenever possible and that dental sedation should be used in preference. Thus, dental surgeons in the UK must now find alternatives to general anaesthesia. Many other countries have adopted American behavioural and pharmacological techniques in order to manage anxious paediatric dental patients and this is the obvious solution to the problems in the UK.

The object of this study was to assess the level of cooperation in children who were undergoing dental extractions under general anaesthesia. This information is of importance because it is towards this group of patients that alternative behavioural management and conscious sedation techniques are aimed.

Method

Consultant anaesthetists in the day-case operating theatre at Glasgow Dental Hospital and School, where approximately 25 children are anaesthetised daily, were invited to evaluate the behaviour of each patient for a 2-week period and to note the number of teeth extracted. The dental hospital offers an all day service for extraction of teeth

Table 1. Frankl behaviour scale.

Rating 1	Definitely negative
☐	Refusal of treatment, crying forcefully, fearful, or any other overt evidence of extreme negativism.
Rating 2	Negative
☐	Reluctant to accept treatment, uncooperative, some evidence of negative attitude but not pronounced, i.e. sullen, withdrawn.
Rating 3	Positive
☐	Acceptance of treatment; at times cautious Willingness to comply, at times with reservation but patient follows direction cooperatively.
Rating 4	Definitely positive
☐	Good rapport, interested in the procedure, laughing and enjoying the situation.

under general anaesthesia in healthy children experiencing dental pain. Children who have medical complications or who require the removal of eight teeth or more are referred to the Royal Hospital for Sick Children for inpatient care. Consequently, subjects in this study are healthy children presenting with a dental emergency and are therefore a self-selected random sample. Information about the patient's age, sex and residential area was also collected.

The Frankl Scale [7] was chosen to evaluate behaviour because it is easy to use and has previously been employed in other studies as one of the criteria for patient selection. The Frankl Scale has four behaviour categories ranging from '1': definitely negative to '4': definitely positive (Table 1).

Results

Of the 200 survey forms, 190 were returned complete; 102 of the children were male (54%) and 88 were female (46%). The mean age was 5 years, (range 1–17 years). Most commonly, one (17.9%) or two (23.7%) teeth were extracted, although the mean number of extracted teeth was 3.66 per patient, with a range of 1 to 23.

Sixty-eight percent of the children (130) lived within the Greater Glasgow Health Board area. Others were from Renfrewshire (12%), Ayrshire (3%), Lanarkshire (3%), Argyll and Clyde (4%) and Stirling (0.5%).

A Frankl scale rating of '3' was allocated in 45.8% (87) of cases; 20.5% (39) of the children were rated '1', 18.9% (36) were rated '4' and 14.7% (28) were rated '2' (Table 2).

Using analysis of variance (ANOVA), a significant correlation was found between the child's behaviour score and age ($p = 0.001$) and between behaviour score and number of teeth extracted ($p = 0.01$). The younger the child and the greater the number of teeth requiring removal, the worse was the perceived behaviour.

Discussion

More than 40% of the children in this sample had only one of two teeth extracted and the majority of the children were given a behaviour rating of '3' on the Frankl Scale. Such a rating indicates cautious acceptance of treatment and a further 18.9% were rated as 'definitely positive'.

The operating theatre is a very different environment from that of the dental surgery and a dental surgeon demands greater attention and cooperation from a conscious paediatric dental patient than that required by an anaesthetist inducing anaesthesia. However, this study has evaluated the behaviour of a specific target group exposed

Table 2. Frankl behaviour ratings for the children included in the study.

Definitely negative	20.5%
Negative	14.7%
Positive	45.8%
Definitely positive	18.9%

to an alien and frightening environment and has shown that 60% of them were reasonably cooperative.

Children in the West of Scotland have a higher incidence of caries than anywhere else in the UK, with the possible exception of Northern Ireland [8]. For many of these children suffering from toothache, the inevitable solution is tooth extraction under general anaesthesia. Such treatment causes psychological trauma and can lead to life-long anxiety about dental treatment. Restorative dentistry is impractical, since this requires a considerable increase in the length of the anaesthetic, therefore teeth are often removed that might otherwise be saved.

Many of the children in the older age groups might well have been responsive to treatment under local anaesthesia and sedation. Such an approach could have reduced the demand for general anaesthesia, so that its use could be reserved for very young children or those who require multiple extractions.

Appropriate training for dentists in sedation, both at the undergraduate and the postgraduate level, is required urgently to encourage a different approach to the provision of dental care for children.

Acknowledgments

The authors thank those members of staff of the Anaesthetic Department of the Western Infirmary and of the Department of Oral Surgery, Glasgow Dental Hospital and School who kindly agreed to participate in this study.

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Diclofenac for analgesia after Caesarean section

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Summary

The analgesic efficacy of a single intramuscular dose of 75 mg diclofenac given after elective Caesarean section was studied in 50 women in a double-blind randomised manner using a patient-controlled analgesia system. The mean 18 h papavaretum consumption of the placebo group was significantly greater (91.4 mg compared to 61.4 mg). Subjective experience of pain and observed sedation were significantly greater in the control group up to 6 h after operation.

Key words

Pain; postoperative, patient controlled analgesia. Analgesics; diclofenac.

Caesarean section is commonly associated with post-operative pain, but may be performed using regional anaesthetic techniques which can be modified to provide analgesia in the postoperative period with advantage [1]. Analgesia after general anaesthesia is usually provided by a parenteral opioid which may produce side effects such as nausea, sedation and respiratory depression. Patients are mobilised early after obstetric surgery [2] and might benefit from the addition of supplementary analgesia, which would reduce their opioid requirements and any drug-related side effects.

Nonsteroidal anti-inflammatory drugs (NSAIDs), such as diclofenac, have been shown to reduce opioid requirements after orthopaedic [3], abdominal [4] and gynaecological [5] surgery and a lower incidence of respiratory depression and a reduction in pain scores have been claimed, with few adverse effects [4]. The analgesic effects of NSAIDs have not been investigated in patients after Caesarean section. The aim of the study was to assess the value of a single dose of diclofenac given intramuscularly after elective Caesarean section performed under general anaesthesia.

Methods

Fifty patients of ASA grade 1 or 2 scheduled for elective Caesarean section under general anaesthesia gave verbal informed consent to this double-blind, randomised, controlled trial. Patients were not studied if they were known to have pre-eclampsia or contraindications to opioids or NSAIDs. Hospital ethics committee approval was obtained. Patients were visited before surgery and instructed in the use of the patient-controlled analgesia system. Premedication of ranitidine 150 mg orally the evening before and on the morning of surgery and 30 ml of 0.3 M sodium citrate were given by mouth immediately before anaesthesia. General anaesthesia was induced with a sleep dose of thiopentone and tracheal intubation was facilitated by suxamethonium 1 mg.kg⁻¹. Anaesthesia was maintained with isoflurane in nitrous oxide and oxygen.

The F_{IO_2} was 0.5 before delivery and 0.3 after. Atracurium 0.4 mg.kg⁻¹ was used to maintain muscle relaxation. After delivery, syntocinon and papavaretum 0.3 mg.kg⁻¹ were given, and atropine 1.2 mg and neostigmine 2.5 mg antagonised neuromuscular blockade after surgery.

Patients were allocated at random to two groups who received either 75 mg diclofenac (group A), or an equal volume of normal saline (group B), intramuscularly before discontinuing anaesthesia. The solutions were drawn up by an independent operator not involved in the study.

Analgesia was provided by a patient-controlled analgesia system (PCAS) (Graseby Medical) set to deliver 3–5 mg boli of papavaretum, according to the patient's weight, with a lockout time of 10 min, and no background infusion. The system was connected to the patient's intravenous infusion by a unidirectional valve, and details of the requests for analgesia were recorded in real time by a dedicated thermal printer (Hewlett Packard 82162-A). Intramuscular antiemetic and additional opioid analgesia were available on demand. Each patient was visited at 6, 12 and 24 h after surgery when the correct use of the PCAS was confirmed and assessments of pain, nausea and sedation were recorded. The patients were asked to complete a 10 cm linear analogue scale (LAS) for pain [6]. Four-point verbal rating scores (VRS) were used to rate nausea (none, nausea, vomiting or repeated vomiting) and sedation (awake, drowsy, rousable or deep sleep). The study was terminated at 24 h or earlier if the patient was mobilised or the intravenous infusion was stopped. A four-point verbal rating scale was used at the end of the study period to rate the overall severity of pain experienced (none, mild, moderate or severe). Specific enquiry about injection site discomfort was made. Results were analysed using the Northwick Park statistics package. Demographic data were compared using Student's unpaired *t*-test; the Wilcoxon rank sum test was used for the LAS data and the Chi-squared test for the sedation and nausea VRS data. A *p* value of < 0.05 was considered to be significant.

Results

Twenty-three patients received diclofenac (group A) and 25 normal saline (group B). Two patients were lost to the study because of missing data from the PCAS printer.

Footnote:

Preparations of papavaretum containing noscopine may no longer be given to women of childbearing age (Ed).

Table 1. Patient data (mean, SD).

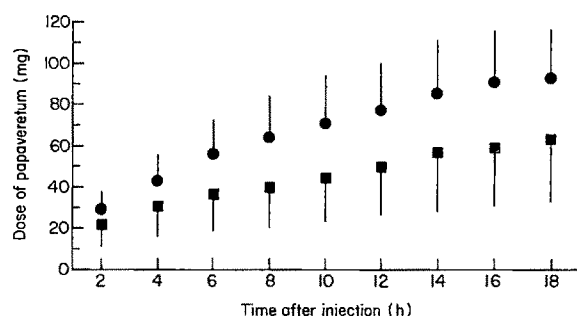
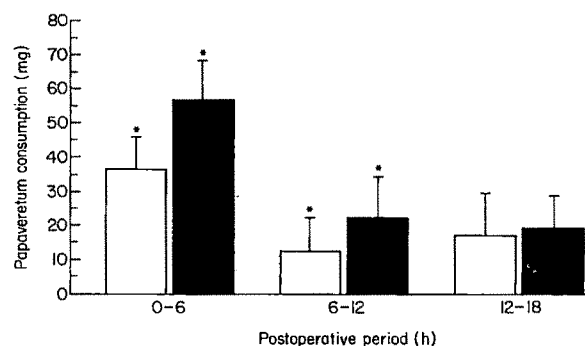
	Group A	Group B
Age; years	26.8 (4.3)	28.3 (5.4)
Weight; kg	72.0 (12.1)	67.2 (9.1)
Height; cm	157.3 (9.0)	158.7 (6.9)

Most patients were mobilised or had stopped intravenous therapy well before 24 h. Demographic data did not differ between groups (Table 1). No patients required additional rescue analgesia and there were no adverse effects to diclofenac. Cumulative papaveretum consumption was significantly greater at 18 h in group B ($p < 0.05$), mean (SD) 91.4 (23.4) mg, than in group A, mean (SD) 61.4 (30.2) mg (Fig. 1). Analysis of papaveretum consumption in 6 hourly epochs revealed a significantly greater consumption in group B compared to group A in the 0–6 h epoch ($p < 0.01$) and in the 6–12 h epoch ($p < 0.05$). There was no difference in papaveretum consumption between the groups in the 12–18 h epoch (Fig. 2). Linear analogue scores for pain were significantly lower in the diclofenac group at 6 h, ($p < 0.05$); the median (interquartile range) score for group A was 0.5 (0.2–2.0) mm and for group B 2.0 (0.1–3.5) mm. There was no difference in the linear analogue scores for pain at 12 h; the median (interquartile range) for group A was 1.2 (0.2–3.6) mm and for group B 1.5 (1.1–3.4) mm. VRS for sedation were significantly lower in the group who received diclofenac at 6 h ($p < 0.05$) (Table 2).

There were no differences between sedation scores by 12 h or between nausea scores at any time between the groups. Two patients in each group commented on mild discomfort at the site of injection, but there was no significant difference between the VRS for the overall severity of pain experienced during the study period. In the diclofenac group, 15 patients rated their pain as mild, and eight as moderate over the study period. Fourteen of the saline group rated their overall pain as mild and 11 as moderate.

Discussion

This study demonstrates that the addition of a single dose of intramuscular diclofenac 75 mg given immediately after Caesarean section reduces papaveretum consumption by one third during the first 18 h, and in the first 6 h equal or superior analgesia was achieved with less sedation when diclofenac was given. These results agree with previous work which demonstrated that diclofenac reduces opioid requirements after hysterectomy and abdominal surgery [4, 5], procedures which represent a similar surgical insult to Caesarean section. However, diclofenac was less effective after cholecystectomy, where an upper abdominal

**Fig. 1.** Mean cumulative papaveretum consumption (mg) in the placebo (●) and the diclofenac (■) groups. Bars represent SD.**Fig. 2.** Mean 6 hourly papaveretum consumption in the diclofenac (□) and the placebo (■) groups. Bars represent SD * $p < 0.05$.

incision may represent a greater insult [7]. Plasma clearance of diclofenac takes about 4 to 5 h after intramuscular injection in healthy volunteers [8] and in our study most of the opioid-sparing effect occurred within the first few hours after the drug was given. This may explain why the reduction in pain and sedation scores were limited to the first 6 h and disappeared after this. We did not administer a further dose of diclofenac, which would have been necessary to continue the analgesia into the second half of our study, because we wished to minimise the number of intramuscular injections given to our patients and the absorption of the oral preparation of the drug could not be predicted so soon after abdominal surgery. There was no reduction in nausea in those of our patients who received diclofenac despite a reduced opioid consumption. This is not unexpected; many other factors have been shown to influence the incidence of postoperative nausea and vomiting in addition to peri-operative opioid [9]. The linear analogue pain scores were low in our study compared to the findings of others who investigated pain relief after Caesarean section [10]. The explanation for this difference is unknown, but all our patients rated their pain, over the whole study period, as mild or moderate and mobilised rapidly. This observation confirms the effectiveness of PCA after Caesarean section, as previously demonstrated [11]. It also suggests that opioid consumption during the study period provided an objective measure of the pain experienced. For this reason the reduced opioid consumption by the patients given diclofenac in our study indicates that this

Table 2. Assessment of severity of sedation and nausea (number of patients per category) * $p < 0.05$.

	Sedation			
	6 h		12 h	
	Group A*	Group B*	Group A	Group B
Awake	17	10	14	12
Drowsy	4	11	4	8
Rousable	2	3	2	2
Asleep	0	1	3	3
	Nausea			
	6 h		12 h	
	Group A	Group B	Group A	Group B
None	12	14	17	16
Nausea	6	9	2	6
Vomiting	5	1	3	0
Repeated vomiting	0	1	1	2

drug has a worthwhile analgesic effect when given after Caesarean section.

It has been suggested that giving NSAIDs before surgery begins may minimise the initiation of pain in the peripheral tissues and enhance their effectiveness as analgesics [12]. We did not give diclofenac until after delivery because NSAIDs are known to induce premature closure of the ductus arteriosus when given in large doses to the mother before delivery. The breast milk of women treated with diclofenac contains extremely small amounts of the drug [13], a quantity not felt likely to represent a hazard to the infants of breast feeding mothers (personal communication, Geigy Pharmaceuticals). We did not attempt to measure diclofenac in breast milk, but there were no problems attributed to it in our mother's infants. We conclude that a single dose of 75 mg diclofenac given intramuscularly has a useful role in the provision of pain relief after Caesarean section performed under general anaesthesia.

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The effect of paracetamol or diclofenac administered before operation on postoperative pain and behaviour after adenoidectomy in small children

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Summary

We compared the effects of rectally administered diclofenac (12.5 mg) with paracetamol (125 mg) on pre- and postoperative behaviour and the need for supplementary analgesia in 44 children scheduled for adenoidectomy (with or without myringotomy). The study drugs were given in combination with diazepam (0.5 mg.kg^{-1}) about 20 min before the children were taken to the operating theatre. On arrival there, the children who had received diclofenac were significantly quieter ($p < 0.05$), easier to handle ($p < 0.01$) and cried less ($p < 0.05$) than those in the paracetamol group. During recovery, children in the diclofenac group needed fewer supplementary doses of intravenous pethidine than those receiving paracetamol ($p < 0.001$). There were no obvious differences between the groups in intra-operative bleeding (as estimated by the surgeon), or in measured blood loss. No postoperative complications became evident. The pre-operative rectal administration of diclofenac for pain relief after adenotomy is safe and effective.

Key words

*Analgesics; diclofenac, paracetamol.
Anaesthesia; paediatric.
Pain; postoperative.*

The use of nonsteroidal anti-inflammatory drugs (NSAIDs) for supplementation of postoperative pain treatment has gained wide acceptance. Because of their mode of action it is preferable that these drugs are given prophylactically before the synthesis of prostaglandins is initiated by tissue trauma. This method has been shown to provide improved analgesia after superficial surgery when compared to postoperative medication [1], although other workers have produced conflicting results [2, 3].

Paracetamol is a widely used analgesic in small children. It lacks many of the serious side effects of opioids and is only slightly sedating. Diclofenac is a well established NSAID which is known to have useful postoperative analgesic properties in adults and which is now available for use in small children.

The aim of the study was to compare, in small children, the behavioural and analgesic effects of rectally administered diclofenac (12.5 mg) with paracetamol (125 mg).

Methods

The study was approved by the Ethics Committee of Tampere University Hospital and written informed parental consent was obtained. Forty-four children, ASA I, weighing 11.0–14.0 kg and scheduled for adenoidectomy (with or without myringotomy) entered the study. Patients were not studied for the following reasons: suspected or manifest bleeding disturbances, allergy to aspirin-like drugs, atopy, bronchial asthma, or a history of serious side effects due to aspirin-like drugs.

Before operation the children were allocated randomly to receive, in a double-blind manner, a rectal suppository of either paracetamol 125 mg (Panadol, Winthrop Ltd) or diclofenac 12.5 mg (Voltaren, Ciba Geigy AG). Both were given with rectal diazepam (0.5 mg.kg^{-1}) (Apozepam, Dumex AL) on the ward 20 min before leaving for the operating theatre. In all cases EMLA cream (Astra Ltd)

was applied beforehand to sites suitable for venous cannulation. On the advice of parents, comforters, drinking bottles, toys etc. were brought with the children to the operating theatre.

Following cannulation of a dorsal hand vein, glycopyrronium (0.004 mg.kg^{-1}) was given intravenously and anaesthesia induced with thiopentone (5 mg.kg^{-1} adjusted to the nearest 25 mg) and suxamethonium (1 mg.kg^{-1}). Anaesthesia was maintained with 67% nitrous oxide in oxygen supplemented with halothane. All patients breathed spontaneously through a Bain coaxial system. At the end of surgery, the inhalational agents were discontinued, the trachea and pharynx suctioned and the patients' tracheas extubated. The time of extubation was recorded. After extubation the patients were transferred to the recovery room where they were kept lying on one side in a 15 degree head-down position to prevent aspiration of secretions. The children left for the ward when they were fully awake, free of pain, and had a stable circulation.

The anaesthetist and the surgeon separately evaluated the degree of salivation at the beginning of the surgical procedure. At the end of surgery the surgeon was asked for his opinion on the extent of bleeding. Intra-operative blood loss was estimated from the increase in weight of swabs and the content of suction bottles.

Pain behaviour was evaluated using seven criteria: alertness, response to handling, anxiety, crying, restlessness, nausea, and breathing pattern. Estimation was on an open scale scoring from 1 to 9 (best to worst). Two nurses evaluated each child: the anaesthesia nurse for the time from arrival of the patient in the operating theatre until transfer of the patient to the recovery room; the recovery room nurse until the patient left the recovery room. Evaluation was performed on arrival at the operating theatre, after insertion of the venous cannula, postoperatively immediately after extubation, on arrival in the recovery room, and at 10 min intervals in the recovery

Table 1. Children's characteristics and duration of surgery, mean (SD).

	Paracetamol (n = 25)	Diclofenac (n = 19)
Age; months	20.7 (6.19)	25.0 (8.98)
Weight; kg	12.2 (10.8)	12.6 (9.61)
Height; cm	85.5 (6.7)	88.4 (4.8)
Duration of surgery; min	21.9 (12.0)	20.3 (6.5)

room until leaving for the ward. Postoperative bleeding complications and other possible side effects were recorded during the first 24 postoperative hours.

Similar methods to those used by parents at home were employed first of all in calming down the child during emergence from anaesthesia. If this did not help, pethidine in increments of 5 mg every 5 min was given intravenously until the child became calm.

Statistical analysis was performed using one-way ANOVA, the Chi-squared test and the two-tailed Student's *t*-test, as appropriate.

Results

Of the 44 children admitted to the study, 25 received paracetamol and 19 received diclofenac. The two groups were similar in respect of age, weight, height and duration of surgery (Table 1).

The mean (SD) estimated blood loss was 9.2 (5.9) ml in the paracetamol group compared with 11.4 (11.2) in those receiving diclofenac. There were also no significant differences between the groups regarding the surgeon's estimate of intra-operative bleeding. No postoperative complications were observed during the stay in the recovery room or later on the ward.

The use of comforters, drinking bottles, etc. and caressing was equal in both groups. Children in the diclofenac group were significantly less restless ($p < 0.05$), easier to handle ($p < 0.01$), and cried significantly less on arrival

Table 2. Childrens' mean (SD) scores (1–best, 9–worst) on arrival in the operating theatre, 10 min and 20 min after arrival in the recovery room, and when leaving.

	Paracetamol (n = 25)	Diclofenac (n = 19)
Alertness		
on arrival	1.9 (2.3)	2.3 (2.9)
10 min	8.0 (1.4)	7.3 (2.4)
20 min	6.6 (2.7)	6.1 (3.4)
when leaving	2.3 (2.6)	1.2 (0.4)
Restlessness		
on arrival	2.9 (3.1)	1.5 (1.4)*
10 min	3.3 (3.1)	3.9 (3.4)
20 min	3.0 (3.1)	2.6 (2.7)
when leaving	1.5 (1.5)	2.4 (2.6)
Response to handling		
on arrival	3.4 (3.0)	1.1 (0.8)**
10 min	5.8 (3.4)	6.1 (2.7)
20 min	5.8 (2.7)	4.9 (2.7)
when leaving	2.0 (2.1)	2.2 (2.4)
Crying		
on arrival	3.1 (3.2)	1.4 (1.7)*
10 min	3.1 (3.0)	5.2 (3.7)
20 min	3.3 (3.1)	3.8 (3.3)
when leaving	1.5 (1.6)	2.6 (2.9)

* $p < 0.05$.

** $p < 0.01$.

Table 3. Supplementary intravenous doses of pethidine (5 mg) in the recovery room.

	Paracetamol (n = 25)	Diclofenac (n = 19)
No. of children requiring pethidine	20	9
No. of doses of 5 mg pethidine	30	20

$p < 0.001$.

in the operating theatre than those of the paracetamol group ($p < 0.05$) (Table 2). No other significant differences in the behavioural criteria were found at any time.

In those receiving paracetamol, 30 supplemental doses of pethidine were needed (in 20 children), whereas those children given diclofenac needed 20 supplemental doses (in 9 children) ($p < 0.001$) (Table 3).

Discussion

It is very difficult to assess pain in very young children. Children in the age range studied here cannot communicate verbally; they are not able to handle abstract concepts, and usually lack experience of painful stimuli. Therefore, one has to rely on altered behaviour for estimation of pain. Different methods and scores are in use to estimate pain-induced behaviour changes in these circumstances [4] but their reliability depends on the familiarity of the investigators with the scoring system. Our nurses are familiar with scoring patients' behaviour because we have used similar scores for many years in this department [5].

The rectal route was chosen for administration of the premedication because intramuscular injection is painful and will increase apprehension and fear. On arrival in the operating theatre the blood diazepam concentrations should have reached a peak together with those of paracetamol ($t_{\max}$ approx. 1.0 h) and diclofenac ($t_{\max} = 0.5$ h) [6, 7].

In animal studies, paracetamol produced dose-dependent central nervous system depression when evaluated by behavioural changes and effects on the EEG [8]. In man, paracetamol may induce mild to moderate drowsiness [9, 10]. Diclofenac, conversely, seems to have a stimulant effect [11]. It may be that the combination of diazepam and paracetamol makes small children more confused and anxious than the combination of diazepam and diclofenac.

We gave diclofenac or paracetamol before operation because there is evidence that this type of drug is best given before tissue trauma to inhibit the activation of the nociceptors [1, 12]. Our findings confirm the results of a recent study after dental surgery [1]. However, the results with other NSAIDs after major abdominal surgery are less convincing. Tigerstedt and colleagues showed that when using indomethacin in adults it takes about 5 h after surgery until differences in opioid medication become significant [2]. In an earlier study using infusions of either diclofenac or indomethacin from just before the induction of anaesthesia, we showed that this regimen had no effect on postoperative pain relief after gynaecological laparotomy [3]. In contrast, the opioid requirements after orthopaedic or general surgery in children aged 1–16 years was less during the early recovery period [13]. These discrepancies may be due to differences in the timing of drug administration and variations in dosage or injection technique.

The hyperalgesic effect of the various prostanoids varies considerably. After a rapid onset, the effect of prostacyclin (PGI_2) only lasts for approximately 30 min, whereas prostaglandins E_1 and E_2 cause hyperalgesia for up to 3 h

after a slow onset [14]. Thus several hours may pass before the maximum effect on pain threshold is reached when using inhibitors of prostaglandin synthesis. This offers a further explanation of why, under different circumstances, pain relief with NSAIDs is achieved at different intervals [12]. In the present study the time interval between application of the premedication and arrival in the recovery room was approximately 50 min.

Unlike the aspirin-like drugs, paracetamol does not inhibit thromboxane A_2 synthesis [15] and therefore does not affect bleeding time [16]. We found no differences in blood loss or bleeding tendency between the two treatment groups and this is in accordance with previous reports [17–20].

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EMLA for postoperative analgesia for day case circumcision in children

A comparison with dorsal nerve of penis block

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Summary

A randomised study was designed to evaluate and compare the analgesic efficacy of EMLA with dorsal nerve of penis block, in children undergoing day case circumcision. The mean (SD) duration of analgesia for EMLA and dorsal nerve block were 0.5 (0.1) h and 6.4 (1.2) h respectively ($p < 0.001$). It is concluded that EMLA is not as effective as dorsal nerve of penis block with regard to postoperative analgesia. No adverse effects were observed.

Key words

Pain; postoperative.

Anaesthetic technique, regional; dorsal nerve of penis block.

Anaesthetics, local; EMLA cream.

Surgery; circumcision.

Circumcision in children is being performed increasingly on a day care basis. It is associated with considerable postoperative pain which may result in restlessness, agitation and bleeding. Several analgesic techniques for circumcision have been described. The use of topical analgesia was first described by Tree-Trakarn *et al.* [1, 2] who reported that the analgesic effects of lignocaine gel, ointment and spray were comparable to that of dorsal nerve block of the penis, with the pain-free period lasting 4–6 h. However, effective analgesia occurred in only 70% [2] and 95% [1] of children and in adults the postoperative pain scores between the topical lignocaine and the placebo groups did not differ significantly.

EMLA cream (a 5% eutectic mixture of lignocaine and prilocaine base) has been demonstrated to produce more effective cutaneous analgesia than each local anaesthetic base given alone in the same total concentration [3]. The greater effectiveness is attributed to the higher concentration of active local anaesthetic bases in the emulsion droplets (approximately 80%) compared with only 20% if lignocaine alone is emulsified [4]. Hence, theoretically, EMLA should provide considerably more effective, potent and prolonged postoperative analgesia than topical lignocaine for circumcision. This study was designed to evaluate the use of EMLA and its efficacy in children for day case circumcision, using an application technique similar to that described by Tree-Trakarn *et al.* [1, 2].

Methods

After informed written parental consent had been obtained, 20 boys (ASA 1) aged between 2 and 10 years, undergoing elective day case circumcision, were randomly allocated to two equal groups. Group A received dorsal nerve of penis block and group B received topical EMLA. The study was approved by the hospital ethics sub-committee. None of the children had a history of drug allergy. No pre-medication was given and EMLA was applied to both antecubital fossae 1 h before intravenous cannulation at

induction. Anaesthesia was induced with propofol (2.5 mg.kg^{-1}) and maintained with isoflurane in nitrous oxide and oxygen. No analgesic supplement was used apart from in the postoperative period.

After induction, dorsal nerve block [5] was performed with an aseptic technique, by the same author (J.J.L.) throughout, using plain bupivacaine 0.5% (1 mg.kg^{-1}). Group B had EMLA 2 g applied over the wound by the surgeon at the end of operation and before surgical dressing. The same EMLA tube was kept with the patient for re-application as required later.

The duration of anaesthesia was noted. The recovery time was recorded as the time from termination of general anaesthesia to the time the child opened his eyes in response to calling his name. Postoperative pain was assessed by the same staff nurse, who was unaware of the treatment groups until the first postoperative analgesia was required. Objective evaluation was recorded at 5 min intervals until discharge using the scoring system shown in Table 1 [6]. The pain-free period was defined as the period from the end of anaesthesia to the time the child started to complain or cry of pain. Postoperative analgesia was administered at a pain score of 6 or more [6]. The treatment group was then revealed for the purpose of investigating the effects of re-application of EMLA. Group A was prescribed paracetamol (120–240 mg orally) and/or codeine (1 mg.kg^{-1} intramuscularly). Group B, after being given a sympathetic explanation, received a further application of EMLA 2 g to the wound and if satisfactory analgesia was not achieved after 10 min, additional paracetamol and/or codeine was prescribed. Any adverse effects were noted.

The children were discharged from hospital when they were comfortable and fulfilled the criteria for discharge. If they were discharged before experiencing pain, the parents were instructed to record the time of onset of pain, the severity of pain using a simple scale (Table 2), and the frequency of analgesic requirements for the first 24 h at home. Group A patients used paracetamol only and group

Table 1. Pain/discomfort scale [6] (reproduced with permission).

Observation	Criteria	Points
Arterial blood pressure	BP $\pm$ 10% pre-operative	0
	BP > 20% pre-operative	1
	BP > 30% pre-operative	2
Crying	Not crying	0
	Crying but responds to tender loving care (TLC)	1
	Crying and does not respond to TLC	2
Movement	None	0
	Restless	1
	Thrashing	2
Agitation	Patient asleep or calm	0
	Mild	1
	Hysterical	2
Posture	No special posture	0
	Flexing legs and thighs	1
	Holding penis or groin	2

B patients were supplied with a new EMLA tube for application 4 hourly as required and were instructed to use paracetamol for additional analgesia.

Data were analysed using the unpaired Student's *t*-test and the Mann-Whitney test.

Results

There was no significant difference between the groups with regard to age, weight, duration of general anaesthesia and recovery time (Table 3). The pain scores for both groups before discharge are shown in Figure 1.

The mean (SD) pain-free period was 6.4 (1.2) h for group A compared with 0.5 (0.1) h for group B ($p < 0.001$). The study was terminated early because of marked differences between groups. One technical failure occurred in a 3-year-old boy who, alone in group A, required paracetamol and codeine and he was not studied. The other nine children remained pain free before discharge. At the initial onset of pain in group A, the severity was described as moderate in five children and mild in four. The postoperative pain scores between group A and B were not compared because different scoring systems were used. This was because a simple pain scale (Table 2) had to be provided for parental use at home.

All children in group B required a second EMLA application (pain score > 6) within 45 min of circumcision. Half of them obtained satisfactory pain relief within 10 min and required no further analgesia before discharge while the remainder needed additional paracetamol. Analysis of the duration of hospital stay and the requirements for paracetamol with or without EMLA during the first 24 h after discharge, did not show any significant differences between the groups. No adverse effects were observed in either group during hospital stay and at follow-up clinics.

Table 2. Parental assessment at home (if patient is discharged before experiencing pain).

Time of first complaint of pain:
Severity: (please tick)
Mild (slight pain; normal activity; no paracetamol needed)
Moderate (reduced activity; paracetamol needed)
Severe (stopped playing; crying)

Table 3. Patient characteristics and clinical data in the two groups studied; mean (SD or range).

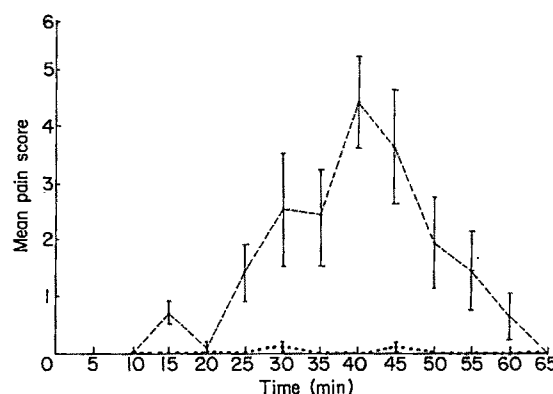
	Group A Dorsal nerve block	Group B EMLA
Number of subjects	10	10
Age; years	6.0 (3.4–10)	5.2 (2.5–10)
Weight; kg	20.8 (4.4)	18.4 (6.4)
Duration of general anaesthesia; min	30.7 (4.7)	31.0 (6.9)
Recovery time; min	10.7 (3.2)	10.5 (5.2)

Discussion

The use of local analgesic techniques in day surgery avoids the adverse effects associated with parenteral opioids and the delay in discharging patients. Dorsal nerve block of the penis has been shown to be a highly effective technique for postoperative analgesia after circumcision [5, 7–11]. However, the procedure may result in haematoma formation and gangrene of the glans penis from accidental vascular puncture has been reported [12]. Systemic toxicity may occur because local anaesthetic is being injected into a highly vascular area and sometimes a block is unsuccessful.

Topical analgesia would appear to offer several advantages for day case circumcision in children. The application technique is simple, requires no special skill and can easily be repeated. Tree-Trakarn *et al.* [1, 2] first described the topical application of local anaesthetics, using lignocaine gel (2%), ointment (5%) or spray (10%) in children undergoing circumcision. All three topical lignocaine preparations were found to be as effective as intramuscular morphine and dorsal nerve block using bupivacaine, with the pain-free period lasting 4–6 h. Topical lignocaine is thought to act by penetrating the mucosal undersurface of the foreskin, the mucous membrane of glans penis, exposed nerve endings and the skin edges which remain after surgery.

Free bases of local anaesthetics penetrate the skin and produce analgesia much faster than do their water-soluble salts [13, 14]. However, it has been demonstrated that the salts of local anaesthetics (such as lignocaine hydrochloride in lignocaine gel) are as readily absorbed as the bases (such as lignocaine ointment and spray, and EMLA) by the mucous membrane and produce blockade within 5 min [15–17]. In the case of lignocaine gel, the duration of effective topical analgesia on the mucous membrane is reported to be 15–30 min [16, 18]. Juhlin *et al.* [4] also commented on the short duration of action of topical

**Fig. 1.** Mean (SEM) pain score before discharge. — — —, EMLA; ·····, dorsal nerve block.

analgesia and this was attributed to the rapid transport away from the tissue to which it had been applied by the underlying blood vessels. Thus, the duration of topical analgesia reported by Tree-Trakarn *et al.* [1, 2] seems surprisingly long and cannot be explained or supported by the known pharmacokinetic profile of topical lignocaine when it is applied to mucous membranes.

In order to achieve adequate topical analgesia of the skin, EMLA must be applied under occlusive dressing for at least 60 min [19, 20]. In contrast, it has been demonstrated clinically that EMLA only needs to be applied to genital mucosa for 5–10 min to achieve adequate topical analgesia for superficial surgery [21–23]. Mucosal topical analgesia has also been shown to diminish in efficacy with increasing application time [22, 23]. This is thought to be due to the decreased rate of transport of the local anaesthetics from the cream into the mucosa when the local anaesthetics in the cream layer closest to the mucosa have been absorbed.

Our data showed that EMLA provided analgesia of less than 45 min duration in the immediate postoperative period, whereas after dorsal nerve block using bupivacaine, the mean duration of analgesia was 6.4 h. Repeated applications of EMLA, though readily accepted by all children, did not produce a satisfactory degree of analgesia. It is concluded that topical EMLA is not an effective analgesic for children undergoing day case circumcision. The short duration of action of EMLA may be partly because it is rubbed off the wound once the child is awake and mobile, and partly because it is rapidly transported away from the area by the rich blood supply.

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A study into the incidence of methaemoglobinaemia after 'three-in-one' block with prilocaine

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Summary

A 34-year-old female patient developed methaemoglobinaemia following a femoral nerve block using prilocaine. The concentrations of methaemoglobin in the blood of the next eight patients receiving this block were assayed, and found to be significantly increased above baseline concentrations, but very significantly less than the concentration in the patient with symptomatic methaemoglobinaemia. Pulse oximetry was of value in making the diagnosis.

Key words

Anaesthetic technique, regional; three-in-one block.

Local anaesthetic; prilocaine.

Complications; methaemoglobinaemia.

Prilocaine is an amide local anaesthetic agent popular for blocks requiring large volumes because of its low potential for systemic toxicity. For this reason it is the agent of choice for femoral nerve block. It is our practice to use this block as the sole anaesthetic technique for muscle biopsies. Following this procedure, one of our patients developed clinical methaemoglobinaemia. Methaemoglobinaemia has long been known as a rare complication of prilocaine administration; however, the incidence of this problem at subclinical levels following femoral block is not known; neither therefore is the margin of safety.

In view of this clinical case, a study was conducted to investigate the incidence and severity of methaemoglobinaemia associated with three-in-one block carried out using prilocaine.

Case history

A 34-year-old female patient was admitted to the Malignant Hyperthermia (MH) Investigation Unit for a muscle biopsy. She was the sister of an MH proband. She was not taking any medication, had no past medical history of note, and had undergone anaesthesia uneventfully on two previous occasions. Physical examination was unremarkable.

Venous access was secured using a 20 G cannula, monitoring with electrocardiograph (ECG) and pulse oximetry (Novamatrix 500) was established, and a femoral nerve block instituted using the three-in-one technique [1] modified by the additional use of a nerve stimulator and pole needle to ensure correct placement [2]. Thirty-six ml of 1% prilocaine (5 mg.kg^{-1}) was injected, and a good quality block, as assessed by loss of cold and pinprick sensation, was established over the anterior and medial thigh. Muscle biopsy was performed satisfactorily and demonstrated the patient to be non-susceptible to MH. During the latter part of the procedure the patient complained of increasing dizziness. The pulse oximeter reading indicated an arterial oxygen saturation falling to 90%. Physical examination revealed mild cyanosis, but no evidence of cardiorespiratory embarrassment. Postoperatively, her oxygen saturation continued to fall to 87%. Peroperative venous blood

sampling for methaemoglobin estimation showed a concentration of 34 g.l^{-1} (OSM3, Radiometer, Copenhagen). Samples taken at 1 and 3 h after institution of the block showed levels of 6% and 6.7% respectively. Methylene blue 70 mg was administered. The pulse oximeter reading transiently fell to 50% before improving rapidly to 95%. The patient was detained in hospital for overnight observation, during which time she felt well. By the following morning her venous methaemoglobin concentration had fallen to undetectable levels, and she was discharged home.

Methods

Ethics committee approval was sought, and informed consent was obtained from eight adult patients (> 18 years of age) scheduled for muscle biopsy under femoral nerve block in the Malignant Hyperthermia Investigation Unit. Patients were excluded who were pregnant or who were known to suffer from methaemoglobinaemia or any other dysaemoglobinaemia, or who were sensitive to amide local anaesthetic agents.

The patients were unpremedicated. ECG monitoring and pulse oximetry (Novamatrix 500) were instituted, and venous access secured with a 20 G cannula. The three-in-one modification of a femoral nerve block was performed as before under nerve-stimulator control [1, 2]. A total volume of 0.5 ml.kg^{-1} of 1% prilocaine was injected. Heparinised blood samples were taken immediately prior to the institution of the block, and at 1 and 6 h after the injection of prilocaine. Methaemoglobin levels were estimated using an OSM3 co-oximeter (Radiometer, Copenhagen). Baseline and subsequent levels were then compared, patients acting as their own controls. Statistical analysis was performed using two-tailed tests; percentage methaemoglobin levels prior to the block, at 1 h and at 6 h were compared using Kruskal–Wallis analysis of variance by rank. The baseline and peak methaemoglobin values were compared using Wilcoxon's rank-sum test for paired results. The peak concentrations for the asymptomatic patients and the initial symptomatic patient were compared using a one-value Student's *t*-test. All results are expressed as mean (95% confidence limits).

Table 1. Methaemoglobin percentage concentration compared with time.

Patient No.	Time; baseline	Time; 1 h	Time; 6 h	(Peak)
1	0.6	2.6	3.4	3.4
2	1.0	3.7	2.9	3.7
3	0.0	1.6	0.5	1.6
4	0.0	1.8	1.1	1.8
5	0.0	0.1	0.0	0.1
6	0.0	1.4	0.4	1.4
7	0.0	0.0	1.1	1.1
8	0.0	0.0	2.5	2.5
Mean values (95% confidence intervals)				
	0.2	1.4	1.49	1.95
	(0-0.48)	(0.46-2.34)	(0.59-2.39)	(1.11-2.79)

Results

The patients' ages ranged from 28 to 66 years and their mean weight was 72.6 (range: 64.2-81.0) kg. The baseline, 1 h and 6 h methaemoglobin levels are shown in Table 1. The increase in mean methaemoglobin concentration from baseline 0.2 (0-0.48) to its peak value of 1.95 (1.11-2.79) was significant at the $p < 0.005$ level, and the trend with time was significant at the $p < 0.01$ level. The mean peak methaemoglobin concentration was significantly lower than the peak concentration in our previous (symptomatic) patient, 1.95 (1.11-2.79) versus 6.7 g% ($p < 0.0001$).

Discussion

Prilocaine has frequently been implicated in the aetiology of methaemoglobinaemia. Methaemoglobin is formed as a result of the oxidation of the iron II (ferrous iron) in haem to the iron III (ferric) form. This naturally occurring process is countered by its reconversion to the ferrous form by the action of the erythrocytic enzyme NADH-dependant methaemoglobin reductase. Methaemoglobinaemia occurs when the capacity of this enzyme system is exceeded. This may be due to a hereditary deficiency in the enzyme system, or as a result of the oxidative stress of any of a number of drugs. Both prilocaine and, more rarely, lignocaine have been implicated; they are both aniline derivatives. Methaemoglobinaemia after prilocaine administration was described by Scott in 1964 and it was shown that not prilocaine but a metabolite was responsible [3]. This metabolite is now recognised as *o*-toluidine [4]. The normal methaemoglobin level is below 1%, and greater than 10% is said to be clinically significant [5]. It is of interest that our initial patient developed clinical methaemoglobinaemia well below this concentration.

Pulse oximetry has previously been described as a valuable if imprecise technique in the detection of methaemoglobinaemia, both in infants [6] and adults [7], but these reports suggest that pulse oximetry may well overestimate the arterial oxygen saturation in the presence of methaemoglobin. Animal work has confirmed that the apparent saturation is unlikely to fall below 84% even in the presence of concentrations of methaemoglobin between 35% and 60% (i.e. true oxyhaemoglobin levels potentially below 40%) [8]. The fact that our first case was symptomatic and clinically cyanosed suggests that her true saturation may well have been substantially poorer than the pulse oximeter indicated. It is of note that this patient was not taking any drugs, so neither pre-existing oxidant stress nor enzyme induction is likely.

Marks and Desgrand have described a case of methaemoglobinaemia following brachial plexus block [9]. However, in their case the dose of prilocaine and of other local anaesthetic agents (which may also, though less commonly, cause methaemoglobinaemia) was unusually

high, owing to a failed and repeated block. They recorded methaemoglobin concentrations in excess of 60 g.l⁻¹. Other cases of methaemoglobinaemia after prilocaine brachial plexus block have been described [10] though surprisingly the problem seems rather less common after intravenous regional anaesthesia. This has been attributed to the slower time-course of systemic prilocaine release after this technique [11].

We do not know of any other reports of methaemoglobinaemia complicating femoral three-in-one block. Our results suggest that our first case developed significantly higher concentrations of methaemoglobin than is common after this technique. However, some degree of methaemoglobinaemia at subclinical levels is usual. In conclusion, in our initial case, as in all of the cases in the recent literature, pulse oximetry was a key tool in making the diagnosis of methaemoglobinaemia. We therefore recommend the use of pulse oximetry, together with an awareness of its limitations, where prilocaine is used for a three-in-one block.

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Administration of vancomycin during cardiopulmonary bypass

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Summary

Vancomycin was given during cardiopulmonary bypass to 12 anaesthetised patients undergoing open heart surgery. Injection of vancomycin 1 g within 60 s via the venous inlet of the oxygenator resulted in a moderate and transient decrease of mean arterial pressure. This minimal reaction may be attributed to dilution of vancomycin by the extracorporeal circuit volume, to the bypassing of the lungs which are a major site of storage of vasoactive substances, or to the maintenance of adequate perfusion flow during cardiopulmonary bypass. The results suggest that the haemodynamic adverse reactions to vancomycin, given as antibiotic prophylaxis, may be decreased by its administration after initiation of cardiopulmonary bypass.

Key words

Antibiotics; vancomycin.
Anaphylactoid reactions.
Cardiopulmonary bypass.

Prophylactic administration of antibiotics in patients undergoing open heart surgery has reduced the incidence of infection. Vancomycin prophylaxis is recommended in patients with known hypersensitivity to the penicillins, and wherever the endemic rate of methicillin-resistant staphylococci is high [1]. However, serious anaphylactoid reactions, including cardiovascular collapse and severe bronchospasm, may follow the infusion of vancomycin [1–5]. Although the incidence and severity of vancomycin-induced reactions may be decreased by using a slow rate of infusion, a number of reports have described the reaction following slow infusions [6, 7].

Vancomycin-induced complications may be potentially life-threatening during the prebypass period in cardiac patients [1, 2, 8]. We have experienced a serious cardiovascular collapse following the administration of vancomycin during the prebypass period in a patient undergoing coronary artery grafting. As a consequence of this we have avoided the administration of vancomycin during the prebypass period and have chosen instead to give it after initiation of cardiopulmonary bypass (CPB). This study was set up to observe the cardiovascular changes associated with this technique.

Method

The investigation was carried out on 12 patients, aged 20–70 years, weighing 50–100 kg, who underwent coronary artery bypass grafting or valve replacement during hypothermic CPB. The investigation was approved by the Institution Research Committee, and informed consent was obtained.

The patients were premedicated with morphine 0.1 mg.kg⁻¹, hyoscine 0.3 mg and promethazine 25 mg given intramuscularly. Anaesthesia was induced with midazolam 0.1 mg.kg⁻¹, fentanyl 50 µg.kg⁻¹ and a mixture of pancuronium 0.1 mg.kg⁻¹ and vecuronium 0.1 mg.kg⁻¹. Following tracheal intubation, ventilation was maintained with 100% oxygen without any inhalational anaesthetic supplementation. Patients were monitored by means of an

electrocardiogram (ECG), a radial artery cannula, and a pulmonary artery catheter.

During CPB, a bubble oxygenator (Bentley-10, American Bentley, Irvine, CA, USA) was used which had been primed with 1500 ml of compound sodium lactate solution. The patients were perfused using a roller pump (Sarns 5000, Ann Arbor, MI, USA) at a constant flow of 2.4 l.min⁻¹.m⁻², and the oxygenator was supplied with an equal flow of 100% oxygen. The temperature was gradually reduced to 27–30°C, and the heart was arrested by a cardioplegic solution (K⁺30 mmol.l⁻¹ at 4°C) after aortic cross clamping.

After about 10–20 min of CPB when the mean arterial pressure (MAP) and reservoir volume had stabilised, vancomycin 1 g diluted in 20 ml of 5% dextrose solution was injected over 60 s into the venous line of the oxygenator. The MAP and reservoir volume were continuously monitored, while the perfusion flow was maintained constant at 2.4 l.min⁻¹.m⁻². MAP and reservoir volume values were recorded every minute from the time of injection over a 10 min period. Changes of MAP and reservoir volume were taken to reflect the effect of vancomycin on the systemic vascular resistance and the capacitance vessels, respectively [9].

The paired *t*-test was used to compare the MAP and reservoir volume values achieved following vancomycin with the control values. A value of *p* < 0.05 was considered significant.

Results

Table 1 shows the mean control MAP value achieved during CPB in the 12 patients and the mean MAP values obtained at every minute from the time of injection of vancomycin over a 10 min period. The MAP significantly decreased from a control level of 76.2 (11.3) mmHg to 52.5 (17.1) mmHg 2 min following vancomycin administration. Recovery to control values was achieved after 6 min.

Table 2 shows the mean control reservoir volume achieved during CPB in the 12 patients and the mean

Table 1. Mean arterial pressure (MAP) in the 12 patients before and after the administration of vancomycin. Values are expressed as mean (SD).

Time; min	MAP; mmHg	% change of MAP*	Significance*
0	76.25 (11.31)	—	—
1	67.08 (9.16)	12.30	< 0.05
2	52.50 (17.12)	31.15	< 0.05
3	57.08 (17.77)	25.14	< 0.05
4	62.50 (17.12)	18.03	< 0.05
5	66.25 (18.23)	13.11	< 0.05
6	69.58 (17.90)	8.75	NS
7	72.92 (18.76)	4.37	NS
8	74.58 (18.88)	2.19	NS
9	73.33 (21.03)	3.83	NS
10	75.83 (20.09)	0.55	NS

0 time, control MAP; NS, not significant.

*Reference to zero minute.

reservoir volume values obtained at every minute following the administration of vancomycin. The reservoir volume gradually decreased within 10 min from a control value of 2.37 (0.96 l) to 1.82 (0.96 l).

Discussion

This report demonstrates the safety of administration of vancomycin after initiation of CPB. The injection of a bolus of vancomycin 1 g over 60 s into the venous inlet to the reservoir of the oxygenator resulted only in a moderate and transient decrease of MAP.

Vancomycin-induced anaphylactoid reaction [1–8] may be attributed to a direct effect on mast cells and basophils that results in their degranulation with subsequent release of various vasoactive mediators; histamine release probably represents the mechanism responsible for hypotension following vancomycin administration in man [10]. Hypotension is the most frequent and the most serious immediate adverse effect associated with the use of vancomycin and the administration of a single dose of vancomycin as antibiotic prophylaxis has been shown to produce a high incidence of severe hypotension in cardiac surgical patients [8].

The minimal and transient decrease of MAP following the rapid administration of a large bolus of vancomycin during CPB may be attributed to dilution by the extracorporeal circuit volume, as well as to bypassing the pulmonary circulation. Adverse cardiovascular responses to drugs such as protamine and vancomycin may be increased by its passage through the pulmonary vasculature as the lungs are a major site of storage of many vasoactive substances, including histamine [11, 12]. Thus, histamine release by vancomycin may be decreased when the lungs are bypassed.

The decreased hypotensive response to vancomycin given during CPB may be also attributed to maintenance of adequate perfusion by the pump, despite the increased venous capacitance as manifested by the decreased reservoir volume. In contrast, during the prebypass period, excessive histamine release does not only decrease systemic vascular resistance but also increases the vascular capacitance and traps large amounts of blood, with a subsequent decrease of the venous return and cardiac output [13]. This will be compounded in the cardiac patient with haemo-

Table 2. Mean reservoir volume (RV) in the 12 patients before and after the administration of vancomycin. Values are expressed as mean (SD).

Time; min	Mean RV; l	% change of mean RV*	Significance* (p value)
0	2.37 (0.96)	—	—
1	2.34 (0.97)	1.27	NS
2	2.22 (1.03)	6.33	NS
3	2.12 (1.04)	10.55	< 0.05
4	2.02 (0.99)	14.77	< 0.05
5	2.01 (1.00)	15.19	< 0.05
6	1.97 (0.97)	16.88	< 0.05
7	1.93 (0.99)	18.56	< 0.05
8	1.90 (0.95)	19.83	< 0.05
9	1.82 (0.97)	23.21	< 0.05
10	1.82 (0.96)	23.21	< 0.05

0 time, control mean RV; NS, not significant.

*Reference to zero minute.

dynamic instability and/or ischaemic heart disease, when cardiovascular collapse may be life-threatening and therapeutic options are narrowed [1, 2].

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Intermittent positive pressure ventilation through a laryngeal mask airway

Is a nasogastric tube useful?

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Summary

A nasogastric tube was used to aspirate air insufflated into the stomach during intermittent positive pressure ventilation through a laryngeal mask airway and a tracheal tube. No difference was found in the amount aspirated between patients with a tracheal tube, a laryngeal mask airway with the nasogastric tube closed or a laryngeal mask airway with the nasogastric tube open, when the nasogastric tube was aspirated at 15 min intervals for the first hour of anaesthesia.

Key words

Equipment; laryngeal mask airway, nasogastric tube.
Complications; gastric insufflation.

Laryngeal mask airways (LMA) are now firmly established in anaesthetic practice. Acceptance has been rapid because of the advantages they have over a facemask. It is easier to maintain an airway with a LMA, they free the anaesthetist's hands, and allow capnography. They can be used either alone or with other aids to overcome a difficult airway. They also allow artificial ventilation of the lungs (IPPV) if it becomes necessary.

There are a number of questions which surround the use of the LMA for IPPV. These concern the isolation of the gastrointestinal tract from the respiratory tract. Payne [1] has demonstrated that in a number of patients with a LMA in place, the oesophagus could be seen through the end of the LMA with a fibreoptic bronchoscope. Brain [2] suggested that this was due to malpositioning of the mask, and that if the mask is properly inserted the oesophagus should not be seen. One possible consequence of inadequate isolation is gastric insufflation. Gastric insufflation has been implicated as a potential factor in reducing diaphragmatic function after operation [3]. It may also be expected to increase the incidence of nausea and have an adverse effect on the gastro-oesophageal sphincter.

The aim of the study was to determine if significantly more air was aspirated through a nasogastric tube in patients who underwent IPPV through a LMA than control groups, and to see if an open nasogastric tube would relieve air insufflated into the stomach.

Methods

The experiment was divided into two parts. Part one compared the amount of air aspirated through a nasogastric tube in patients undergoing IPPV via a tracheal tube with those undergoing IPPV via a LMA. Part two compared two groups, both undergoing IPPV through a LMA with a nasogastric tube in place. One group had the nasogastric tube left open and the other closed.

Approval was obtained from the Hospital Ethics Committee. After giving informed, written consent, 50 fasted patients, who had been classified as ASA 1 or 2 undergoing peripheral surgery, were randomly allocated to one of three groups.

Ten patients were allocated to group 1, 20 to group 2 and 20 to group 3. All patients were premedicated with temazepam and anaesthesia was induced with propofol 2.5 mg.kg⁻¹ and atracurium 0.3 mg.kg⁻¹. The patients' lungs were gently ventilated by facemask with N₂O/O₂ and enflurane 1% for 3 min. A 16 FR single lumen nasogastric tube was inserted and its position checked by injecting 2 ml of air and listening over the stomach. Initial gastric contents were then aspirated. A three-way tap was placed on the end of the nasogastric tube of the patients in groups 1 and 2 and closed off to the patient.

The tracheas of group 1 patients were then intubated, groups 2 and 3 had a LMA inserted. The lungs of all patients were ventilated to normocapnia using an Ohmeda 7000 ventilator, with the tidal volume adjusted to keep the airway pressure less than 20 cmH₂O.

The nasogastric tube in all groups was aspirated with a 20 ml syringe at 15 min intervals for the first hour. In groups 1 and 2, the three-way tap on the end of the nasogastric tube was closed off to the patient after each aspiration. If no air or fluid was aspirated at an interval, 2 ml of air was injected through the nasogastric tube to clear any possible blockage, and aspiration was attempted again. Parametric data were analysed using Student's *t*-test, nonparametric data using the Mann-Whitney *U* test.

Results

Forty-five patients were studied. Three patients were withdrawn because the operation was unexpectedly shortened and in two patients the nasogastric tube could not be placed in the stomach with confidence.

Patients were similar in age and weight (Table 1), and there was no significant difference in the respiratory variables between the three groups (Table 2). All patients were ventilated with airway pressures below 20 cmH₂O, and a high proportion of patients in groups 2 and 3 had an audible leak around the LMA (Table 2).

There was no significant difference in the amounts of air aspirated at each interval between groups 1 (tracheal tube) and 2 (LMA with nasogastric tube closed) or between groups 2 and 3 (LMA with nasogastric tube open). The

Table 1. Demographic data.

Group (n)	1.0 (7.0)	2.0 (20.0)	3.0 (18.0)
Age; years (SD)	40.0 (21.0)	47.0 (20.0)	37.0 (20.0)
Weight; kg (SD)	75.0 (3.5)	67.0 (12.0)	71.0 (14.0)

Table 2. Respiratory parameters.

Group (n)	1 (7)	2 (20)	3 (18)
Tidal volume; ml (SD)	714.0 (124.0)	662.0 (93.0)	691.0 (121.0)
Minute volume 1 (SD)	6.7 (1.7)	6.8 (1.8)	7.1 (2.1)
Airway pressure; cmH ₂ O (SD)	18.0 (2.0)	19.0 (2.1)	17.0 (4.2)
Leak n (%)	0	9.0 (45.0)	8.0 (48.0)

Table 3. Volume of air aspirated from the nasogastric tube in groups 1 and 2. Volumes are mean ml (SD) aspirated at each 15 min interval.

Time (min)	Group 1 TT; ml (SD)	Group 2 LMA; ml (SD)	p value
15	2.57 (3.3)	2.85 (5.9)	0.88
30	0.3 (0.8)	1.65 (3.0)	0.07
45	0.29 (0.76)	0.85 (2.0)	0.31
60	0.29 (0.8)	0.2 (0.89)	0.81

TT, tracheal tube; LMA, laryngeal mask airway.

Table 4. Volumes of air aspirated from the nasogastric tube in groups 2 and 3. Volumes are mean ml (SD) aspirated at each 15 min interval.

Time (min)	Group 2 LMA with nasogastric tube closed; ml (SD)	Group 3 LMA with nasogastric tube open; ml (SD)	p value
15	2.85 (5.84)	3.00 (6.65)	0.94
30	1.65 (3.0)	1.33 (3.55)	0.77
45	0.85 (2.0)	2.11 (8.22)	0.53
60	0.2 (0.89)	0.39 (0.85)	0.51

Table 5. Frequency distribution of total volumes aspirated in groups 1 and 2.

Total volume (ml)	0-20	20-40	> 40
Group 1 (TT) n = 7(%)	7 (100)	0	0
Group 2 (LMA) n = 20(%)	18 (90)	2 (10)	0

For abbreviations, see Table 3.

Table 6. Frequency distribution of total volume aspirated from groups 2 and 3.

Total volume (ml)	0-20	20-40	> 40
Group 2 (LMA with nasogastric tube closed) n = 20 (%)	18 (90)	2 (10)	0
Group 3 (LMA with nasogastric tube open) n = 18 (%)	17 (94)	1 (6)	0

volumes aspirated in all groups were small and decreased with time (Tables 3 and 4).

The total volume aspirated did not exceed 40 ml in any group. One patient in group 2 and two in group 3 aspirated a total volume of more than 20 ml, but less than 40 ml (Tables 5 and 6).

Discussion

There is no readily applicable gold standard for measuring air insufflated into the stomach during IPPV. This study assumes that if a nasogastric tube were to be useful in preventing gastric insufflation, it could also be used to measure the amount of air in the stomach. Inaccuracy in the amount aspirated should be the same in all groups. The aim of the study was to demonstrate a difference between groups if one existed, not to document the amount of gastric insufflation which may have occurred.

If gastric insufflation was occurring in the patients undergoing IPPV through a LMA, insufflated air should have accumulated in the stomachs of patients in group 2. Also, the air insufflated in the patients in group 3 should have escaped through the nasogastric tube, which was open to the atmosphere. The absence of a significant difference between these two groups suggests that either the nasogastric tube was not allowing insufflated air to escape, or gastric insufflation was not occurring. The small volumes of air aspirated and the decreasing volumes with time support the latter. In either case, the nasogastric tube was performing no useful function.

It is still possible, however, that air was insufflated into the stomach of these patients, but aspiration was not possible through the nasogastric tube. It is possible, and even common in some circumstances, for a nasogastric tube to become blocked. In this study, if no air or fluid was aspirated at any interval, 2 ml of air was injected into the nasogastric tube to unblock it and further aspiration attempted. It was assumed that if a significant amount of air was in the stomach, this manoeuvre would have allowed its aspiration.

It is possible that a nasogastric tube would increase the likelihood of gastric insufflation. The absence of a difference in the amount of air aspirated between patients in groups 1 and 2 mitigates against this. The incidence of leak around the LMA in groups 2 and 3 was higher than reported in other studies [4]. This was most likely due to the nasogastric tube *in situ*, which did not allow a tight fit between the LMA and the posterior pharyngeal wall. Ventilator settings were adjusted to minimise the leak, but it should be noted that if airway pressures are maintained below 20 cm, leaks are unusual in patients without a nasogastric tube.

In adults, less than 40 ml of air in the stomach is considered physiologically insignificant. Intra-gastric pressure does not rise until at least 50 ml is injected into the stomach of small animals. [5]. None of the patients in this study had more than 40 ml aspirated in total. Gastric volumes of air are therefore likely to be a small, although the accuracy of this technique in determining the amount of air insufflated is not established.

In summary, this study suggests that a nasogastric tube is of no value when placed prophylactically in normal patients undergoing IPPV through a LMA. Because of the known morbidity associated with placing a nasogastric tube, the possibility that it may adversely affect the lower oesophageal sphincter, and the increased incidence of leak around the LMA when the nasogastric tube is in place, use of a nasogastric tube is not recommended in this group of patients. Because of the small numbers involved in this study, it is possible that significant gastric insufflation may

occur in a small proportion of patients and vigilance by the anaesthetist is therefore always necessary.

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Postoperative oxygenation in the elderly following general or local anaesthesia for ophthalmic surgery

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Summary

Peripheral oxygen saturation was recorded on the pre-operative night (between 2400 and 0600 h), the immediate postoperative period (first 60 min) and the first postoperative night (2400 to 0600 h) in 18 elderly patients aged 70 years or over presenting for elective ophthalmic surgery. Nine patients had surgery performed under general anaesthesia employing muscle relaxants and controlled ventilation and nine under local anaesthesia using a peribulbar block. The median (interquartile range) percentage of time during which the patients had an oxygen saturation of less than 90% was 0 (0–0.2) and 0.04 (0–0.4) on the pre-operative night, 0.7 (0–1.4) and 0.3 (0–1.2) in the immediate postoperative period, and 0.05 (0–0.16) and 0 (0–0.3) on the postoperative night in the general and local anaesthesia patients respectively. There were no significant differences between general and local anaesthesia in respect of these data and the overall incidence of significant desaturation was low. The present study could not demonstrate any adverse effect of general anaesthesia on oxygen saturation in patients undergoing minimally invasive surgery.

Key words

*Anaesthesia; geriatric.
Anaesthetic techniques, regional; peribulbar block.
Hypoxia.*

Various studies have demonstrated a decrease in peripheral oxygen saturation (SpO_2) in the period following anaesthesia and surgery [1–4]. The important factors in its causation may be general anaesthesia, the type of surgery, ability to breathe effectively and the method of postoperative analgesia. It is also thought that age has an important influence [2, 3]. There is, however, little information available about the effects of anaesthesia itself on elderly patients undergoing minimally invasive surgical procedures which do not impede breathing or require strong analgesics. The present study was designed to assess postoperative oxygenation in this population using ophthalmic surgery under local or general anaesthesia as a model.

Methods

Eighteen ASA grade 1 or 2 patients (aged 70 years or over) scheduled to undergo lens extraction were included in the study with their informed consent and ethics committee approval. Patients with obesity, renal or hepatic impairment, chronic bronchitis, asthma, or who were receiving any sedative medication or acetazolamide were not studied. Patients were randomly allocated to receive either a local (LA) or general (GA) anaesthetic. Continuous recordings

of oxygen saturation were made on the night before surgery on all patients using a Datex Satlite oximeter employing a finger probe linked to a microcomputer for data storage. Recordings of oxygen saturation were made every 10 s and stored so that 360 readings were collected for every hour. Measurements took place between 2200 and 0600 h to suit ward routine. The period up to 2400 h was used to allow the patients to become accustomed to the probe, and data were analysed only between 2400 and 0600 h. One of the investigators (GMcC) was present during the recording of all the SpO_2 data.

No night sedation was allowed during the study period and premedication consisted of paracetamol (1.0 g) 90 min before operation. The general anaesthesia group received thiopentone 2–4 mg.kg⁻¹, fentanyl 1 µg.kg⁻¹, and vecuronium 0.08 mg.kg⁻¹, followed by ventilation to normocarbapnia with 66% nitrous oxide in oxygen and 1.5 MAC halothane (age adjusted). Residual neuromuscular block was antagonised with neostigmine 40 µg.kg⁻¹ and glycopyrronium 10 µg.kg⁻¹ and antagonism confirmed by the absence of apparent fade to a tetanic stimulus. Local anaesthesia was instituted by peribulbar block using 7 ml of 2% lignocaine, 3 ml of 0.5% bupivacaine with 1:200 000 adrenaline and 150 units of hyaluronidase. No sedation was given to

Table 1. Physical characteristics of patients and duration of operation. Values are mean (SD).

	General anaesthesia	Local anaesthesia
<i>n</i>	9	9
Age; years	75 (4.2)	77 (4.5)
Weight; kg	68 (9.5)	67 (10.5)
Sex; m/f	3/6	3/6
Duration of procedure; min	49 (13.1)	43 (13.0)

patients undergoing surgery using regional anaesthesia but they received oxygen by a loose fitting Hudson mask at $6\text{ l}\cdot\text{min}^{-1}$ for the duration of their surgery. All patients received oxygen for 15 min at the end of surgery before transfer to the post-anaesthesia recovery ward. No supplementary oxygen was administered postoperatively unless the SpO_2 decreased to less than 85%, the point at which the oximeter triggered an alarm. SpO_2 was recorded for the first postoperative hour and then during the first postoperative night in the same way as on the pre-operative night. Paracetamol was given postoperatively, if required.

The percentage of time during which the patients were significantly desaturated was calculated for each of the three periods and expressed as median values and their interquartile ranges. Significant desaturation was defined as an SpO_2 of less than 90% lasting 20 s or more. The results were tested for statistical significance using the Wilcoxon signed rank test and the Mann-Whitney *U* test.

Results

The groups did not differ significantly in age, weight or duration of surgery (Table 1). The data obtained from two patients (1 GA, 1 LA) were not analysed statistically (see below).

The median oxygen saturations recorded in each patient

Table 2. Individual peripheral oxygen saturation values for each observation period. Values are expressed as medians and interquartile ranges.

General anaesthesia		
Pre-operative night	Immediate postoperative period	Postoperative night
97 (97–97)	95 (94–95)	96 (95–96)
94 (94–95)	95 (94–96)	95 (95–96)
94 (94–95)	95 (94–97)	95 (95–96)
94 (94–95)	95 (94–96)	95 (94–95)
96 (95–96)	97 (96–97)	96 (95–96)
96 (95–96)	97 (96–97)	96 (95–96)
96 (96–97)	95 (94–96)	95 (94–96)
97 (95–96)	95 (93–96)	95 (94–96)
95 (95–96)	95 (93–96)	93 (93–94)*
Local anaesthesia		
94 (95–95)	96 (95–97)	96 (95–96)
95 (93–95)	96 (95–97)	96 (95–97)
94 (93–95)	93 (91–94)	93 (92–94)
95 (93–95)	95 (95–96)	97 (96–97)
95 (94–96)	95 (94–96)	96 (96–97)
97 (97–97)	97 (96–97)	97 (97–97)
94 (93–94)	95 (94–96)	94 (93–94)
96 (95–96)	95 (94–95)	95 (95–96)
92 (91–93)	92 (91–93)	93 (92–94)**

*Incomplete recording on postoperative night.

**Temazepam on preoperative night.

Table 3. Duration of desaturation (% time)*.

	Median	Interquartile range
Pre-operative night		
GA	0	0–0.2
LA	0.04	0–0.4
Immediate postoperative period		
GA	0.7	0–1.4
LA	0.3	0–1.2
Postoperative night		
GA	0.05	0–0.16
LA	0	0–0.3

*Based on eight patients in each group.

GA, general anaesthesia; LA, local anaesthesia.

are given in Table 2. There were no significant differences in these either within the groups for the three time periods or between the groups at each time period. The percentage of time spent with significant desaturation was very low and without any significant difference between the groups.

The patient whose results were excluded from the GA group was a woman aged 76 years, who developed postoperative confusion. She had a median (interquartile range) oxygen saturation on the pre-operative night of 95% (95–96%) with no episodes of saturation less than 90%. In the recovery ward her median saturation was also 95% (93–96%) with only 0.5% of the first hour spent at a saturation of less than 90%. In her case, oxygen saturation measurements were only possible until the development of her confusion when she would no longer tolerate the finger probe. As a result, she had only a 2 h observation period on the postoperative night. The median saturation during this period was 93% (93–94%) with 0% of this time spent at a saturation of less than 90%.

The second patient was an 84-year-old woman in the LA group who was not included in the analysis because she was given temazepam 10 mg inadvertently on the pre-operative night. She had a median saturation of 92% (91–93%) with 10% of the pre-operative night spent at a saturation of less than 90%. In the immediate postoperative period her median saturation was 92% (91–93%) with 1.5% of the time spent at a saturation of less than 90%. During the postoperative night, without sedation, her median saturation was 93% (92–94%) with only 0.2% of the night spent at a saturation of less than 90%.

Coincidental findings in the GA group were urinary retention in one patient and vomiting in three other patients between 6 and 24 h after surgery. No complications were observed in the LA group.

Discussion

Ageing is associated with a diminished pulmonary reserve and deterioration in the control of breathing, which may lead to hypoxia in the postoperative period particularly when compounded by the effects of anaesthesia, painful surgery and postoperative analgesia [5–10]. However, the incidence of episodes of desaturation in both the immediate postoperative period as well as the postoperative night was low in the present study even in patients receiving general anaesthesia. This is in contrast to what has been reported in elderly patients undergoing orthopaedic or body surface operations where postoperative PaO_2 within the first hour after anaesthesia was predicted to decrease with age [11]. Differences in the nature of surgery, occurrence of postoperative pain, as well as premedication and the dose and type of opioids used may explain their findings. Ophthalmic surgery where these factors can be avoided

may represent a better model for the study of effects of anaesthesia alone.

We did not measure the percentage of time our patients were actually asleep in order to minimise any disturbance, but instead looked at hypoxia as an endpoint for assessing the significance of the interaction of age and sleep. Confirmation of sleep by additional apparatus would only have increased the amount of disturbance and sleeplessness and could possibly lead to hypoxia on subsequent nights [12]. It is interesting to note that the presence of the pulse oximeter probe was considered by one patient to be a potential disturbance to sleep on the pre-operative night, and led to the inadvertent administration of sedation by the nursing staff. This problem has been found by other workers, where patients declined to take part because they felt that the measurement might interfere with their sleep [13]. Night sedation with benzodiazepines to overcome this problem may itself produce desaturation, as was shown by one patient in the present study and as has been reported by others [14]. Benzodiazepines, in addition, reduce the tone of the upper airway which can increase any tendency to hypoxia [15]. Occurrence of any hypoxia was therefore chosen as the endpoint for assessing the significance of the interaction of age and sleep.

A limitation of the present study may be that it assessed the oxygen saturation only on the first postoperative night. It has been established that episodes of desaturation can occur for several days postoperatively, because of a tendency for rebound increase in the amount of rapid eye movement (REM) sleep on the third postoperative night. This may, however, be due to the use of morphine analgesia [1] which suppresses REM sleep so that a rebound increase occurs when morphine is withdrawn. This was, however, not a factor in our study because we chose a patient model specifically to isolate the influence of anaesthesia alone on oxygenation, and not premedication or postoperative analgesia. Furthermore a prolonged volunteer study of polysomnographic recordings in young subjects before and after 3 h of isoflurane anaesthesia without surgery indicated that the effects of anaesthesia on sleep were confined to the first postoperative night and consisted of a moderate suppression of slow wave sleep (stages 3 and 4) without any effect on REM sleep [16]. Moreover, in a study of patterns of oxygenation after thoracotomy other workers found the worst patterns on the first postoperative night [17]. We cannot rule out the possibility that saturation values might not be worse in our patients on subsequent nights, because of disturbances in the quality of sleep. However, in this study unless complications developed patients were often discharged the next morning making extended observation impossible.

In conclusion, using ophthalmic patients as a model to minimise any desaturation induced by the site of surgery or postoperative analgesia, we found a very low incidence of significant desaturation in elderly patients pre-operatively, in the immediate postoperative period and on the first postoperative night. This was irrespective of whether patients had received local or general anaesthesia. These findings serve to emphasise the importance of the site of

surgery and analgesia as factors in postoperative hypoxemia.

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Caudal anaesthesia in the Shy-Drager syndrome	1100	The role of the laryngeal mask in prehospital trauma care	1108
<i>F.A.R. Gomesz, FFARCS and M. Montell</i>		<i>M.B. Walker, FRCAnaes</i>	
Nutricath insertion—a hazard	1100	Laryngeal mask airway and low flow anaesthesia	1108
<i>K. Barclay, FRCAnaes, R. Falconer, FFARCS and P. Latto, FFARCS</i>		<i>M.R.W. Stacey, FRCAnaes and A. Shambrook, FRCAnaes</i>	
<i>R.J. Panrucker</i>	1101	Nil by mouth after midnight	1108
Equipment deadspace in paediatric breathing systems	1101	<i>J.R. Maltby, MD and L. Strunin, FRCAnaes</i>	
<i>L.M. Fox</i>		<i>A.J. Matthews, FRCAnaes and R.J. Coultas, FRCAnaes</i>	1109
		<i>R. Armstrong, FFARCS</i>	1109
		<i>S. Phillips, FRCAnaes and S. Hutchinson, FRCAnaes.d</i>	1110

Patient-controlled analgesia: a serious incident

My attention has been drawn to the recent article by Drs Grover and Heath (*Anaesthesia* 1992; 47: 402–4). Unfortunately it contains several factual inaccuracies which I would like to correct.

The Bath Institute of Medical Engineering (BIME)

undertakes the evaluation of infusion devices on behalf of the Medical Devices Directorate (MDD) of the Department of Health. Prior to a full evaluation of any infusion device it is subjected to a brief assessment at BIME, and this may be conducted at any time. Therefore a

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brief assessment is a prelude to a full evaluation and not a full evaluation in itself. Brief assessments *are* brief and do not look at the infusion device in detail, and as such there are no *pass* or *fail* criteria involved.

The Bionica MDS 110 PCA infusion pump was subjected to a brief assessment in June 1990. The brief assessment report was sent to me and to the then UK supplier (International Medication Systems UK Ltd) in confidence on 25 June 1990 *after* the incident described in the case report. A section of this report read: 'The pump was not disassembled and only the external mechanical design was assessed. The only point of criticism was the syringe plunger clamp and drive mechanism. It was noted that the syringe plunger was a slack fit in the clamp and the latter extended only partially over the plunger. This could contribute to backlash in the drive and might be the cause of the inconsistent bolus delivery. It is recommended that the clamp be extended over the whole of the syringe plunger and the clamp be a tight fit.' So it follows that the brief assessment *did* detect the possibility of disengagement of the syringe from the drive mechanism.

On the strength of the brief assessment report and the report of the adverse incident (dated 19 June 1990) MDD, with the full cooperation of the UK supplier, arranged to have all known Bionica MDS 110 PCA pumps removed from use. Known users and locations were notified of this action by telephone on the 4 July 1990 followed up by letter on 6 July 1990. All known pumps were removed from use by 13 July 1990.

Unfortunately, in late February 1991 MDD discovered that there might have been pumps still in use which had been placed by a previous supplier. Doubts about their location necessitated the issue of the Hazard Warning Notice mentioned in the article. Thus some statements in this paper are somewhat misleading when consideration is given to the points as stated above.

I should also like to point out that we have already developed the technical protocols for the evaluation of PCA pumps, and once we have decided how to handle the user evaluation then we can begin the full evaluation of these devices.

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N. JENNINGS

A reply

Thank you for the opportunity to reply to Mr Jennings' letter. It appears to us that he had confused the two examinations of the Bionica pump that were carried out.

The brief assessment referred to in our article was one performed by the Bath evaluation centre at the request of International Medication Systems (IMS) prior to offering the Bionica pump for clinical evaluation in UK. We have not seen a copy of this assessment, but were assured by IMS that no safety implications were apparent from it. The quotation in Mr Jennings' letter appears to be from *this* assessment and does not allude to the possibility of disengagement, merely of slackness which might lead to bolus inaccuracy. The report that correctly identified the problem was indeed undertaken *after* the incident reported by us (which took place on 19 June 1990) and was carried out on the actual pump involved in the incident. IMS had already acted to suspend use of the pumps within 24 h of the incident. We were informed of the contents of the report by letter dated 4 July 1990 from Mr A. A. Baker, Senior Engineer, Medical Devices Directorate. His letter stated 'The above pump was tested at the Department and no fault could be found in terms of flow rate. The pump was run through its self check, at different flow rates, bolus sizes and lock out periods and appeared to operate correctly. It was noticed, however, that when the syringe plunger was well out of the syringe barrel (7–10 ml remaining in the syringe) that the plunger was not positively held by the pump. Tipping the pump would lead to the plunger becoming disengaged from the drive. This is a problem that has been seen by the Department on other pumps and has resulted in syphoning of fluid into the patient.'

As acknowledged in our article, we are grateful to Mr Baker and to Mr Broughton of IMS for helpful comments during drafting of the article; these did not include any indication that we had made inaccurate or misleading statements. We are delighted to hear, 2 years after the incident, that some progress is being made towards the much needed evaluation of PCA equipment. May we take this opportunity of offering any help required with user evaluation.

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M.L. HEATH
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Psychomotor recovery following day-care anaesthesia

We read with interest the article by Marshall *et al.* (*Anaesthesia* 1992; 47: 461–6) on recovery characteristics using isoflurane or propofol for maintenance of anaesthesia. This is not the first study on this subject; our own results were not very different from theirs [1], although somewhat in contrast to those of Korttila *et al.* [2]. We agree that lack of standardization of psychomotor tests leads to difficulty in interpretation of results and perhaps this problem should be addressed more closely.

The problem of 'learning' associated with psychomotor tests is quite genuine and although the authors are correct in saying that a control group should be included when studying psychomotor function, we disagree with the interpretation of their results. Firstly, the statistical significance has been calculated from pre-operative baseline values and not with the control group at the different time periods. This would mean that tests, such as the simple reaction time, which have a 'learning' effect may mask significance even though there is a significant difference. For example,

when looking at the data provided in Table 4, the 3 h test in all the groups (except control) shows no significant difference from pre-operative baseline values. However, there could be a significant difference if these values were compared with the control values at 3 h rather than with pre-operative values. That is to say, the learning effect, which is obvious in the control group, is missed in the other groups and the results misinterpreted as '... the control group therefore provides an invaluable indicator of a short-fall in performance which, in the patient groups, shows as a failure to improve on baseline scores'. A two-way analysis of variance (ANOVA) looking at both time and treatment may also have been a useful statistic if the data were distributed normally, otherwise the Friedman's test could have been used instead of the Wilcoxon Rank Sum test. A similar argument applies to the results of the Salford tracker scores where the authors have again shown a significant 'learning' effect in the control group, but not in the other groups. If the results at various time periods had

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been compared to the control group instead of baseline pre-operative scores, the results may have been quite different. Also, in view of the 'nonsignificant' difference between the two groups, perhaps a power function of the test should have been done to see whether the number of patients studied was adequate to reach to any conclusions [3].

Papaveratum may affect psychomotor performance and since there was a difference in the number of patients given papaveratum in the different groups, this may have affected the results. It also appears (although it is not clear from the results) that those patients who were unable to perform the psychomotor tests have been excluded when giving the median values of the simple reaction time. If indeed this is true, the results may be quite different if these patients were also included when calculating the median values. Besides, since the number of patients who could not perform the test was quite large (up to 36%) at 1 h and in two groups even at 3 h (up to 20%), it is difficult to conclude either that there was no significant difference from baseline values at 3 h in any group, or that there was no difference between the groups!

In summary, one must be careful in interpreting results obtained from studies where psychomotor performance is measured using tests associated with 'learning'. A new test, the perceptive accuracy test, has, in this respect, not been found to have any learning effect [1, 4] but more studies are needed using this test before any conclusions can be made.

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Resistance to gas flow in heat and moisture exchangers

I read with interest the article on the comparison of heat and moisture exchangers (HMEs) (*Anaesthesia* 1992; 47: 414–20). I agree that the resistance to gas flow is a key determinant in the use of HMEs. However, the methodology used in this study must be questioned. I evaluate HMEs for the Department of Health, the results of which are published in the *Evaluation* series.

The authors quote 0.5 kPa at a gas flow of 60 l.min⁻¹ as the limit of acceptability for the pressure drop across a HME from the draft International Standard for HMEs [1]. However, the test method employed by the authors to determine the pressure drop under wet conditions differs from that described in the draft standard. This requires that the HME is connected to a patient model expiring fully saturated gas at 34 °C. The model is then ventilated for 24 h. This method has the advantage of not requiring the addition of an arbitrary amount of water to the HME, as described in the article, but, rather, it closely simulates the clinical situation. Using this method I found that, contrary to the article in question, all the devices I tested in common with the authors [2–4] complied with the Standard in both the dry and wet state. These results are in line with those of other authors [5, 6]. This test arrangement also allows an indication of the moisture conserving performance of the HMEs to be made, information that was notable by its absence in the article. A low moisture output has been implicated in causing tracheal tube occlusions [7–10].

The effect of the resistance to gas flow of HMEs has also been investigated *in vivo* [11]. No significant increase in resistance was found after 12 h of use. Buckley has, however, described a large increase in pressure drop across the Pall filter due to condensation within the filter body [12]. It was concluded that the positioning of the filter was an important factor. In the light of the study in

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A reply

We have read the comments of Dr Gupta and colleagues but feel that their suggestions, which include comparing unanaesthetised controls directly with postoperative patients, may lead to more, rather than less, potential for ambiguity in the interpretation of the results. Testing a disparate group of patients in the clinical setting of day case surgery has inherent difficulties; if the perception accuracy test, described by the authors, has indeed no learning effect it may be a valuable addition to this area of research.

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R.M. JONES

question, this perhaps needs to be re-emphasised. If the HME is positioned vertically at the highest point in the breathing system, excessive water will not collect within the HME body, and therefore an increase in pressure drop, as noted by the authors, will not occur.

The authors suggested, but did not test, the hypothesis that large-pore devices, such as the Filt-a-Therm and Hygrobac, have a higher potential for blockage if such a filter is hanging down and collecting secretions. I would suggest, however, that the hydrophobicity of the Pall filter would exacerbate this problem, not reduce it, by preventing the breakthrough of water if an obstruction occurred. Such an obstruction would allow inspiration, but prevent expiration. Thus the breakthrough of condensate in a large-pore device may be considered to be an advantage, rather than a hazard as proposed in the article, in that it would prevent damage to the patient's lungs by high pressures if obstruction occurred.

I do not agree with the authors that HMEs are used increasingly as filters. The reference to this [13] was published 4 years before Chalon *et al.* employed the Pall filter as a HME [14]; surely a case of filters being used increasingly as HMEs. Indeed, with the current plethora of devices incorporating bacterial/viral filters, it is difficult to imagine why 'pure' HMEs would still be employed as filters if the prevention of bacterial/viral contamination was considered essential.

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A reply

Thank you for the opportunity to reply. Much information already exists on the moisture conserving performance of

heat and moisture exchangers (HMEs) [1]. Our interest was primarily in their filtration and airflow characteristics under different conditions. The draft hydrophobicity test is useful for assessing the performance of devices in a simulated humidified environment. However, patients in the intensive care unit may be producing copious secretions which may then enter the filter housing. We believe that hydrophobicity is a key element to adequate filtration and this study highlighted differences between filters which are not apparent from the draft test and which have implications regarding safety.

Correct positioning of a filter is obviously important. Buckley described the use of the Pall filter with a heated water bath humidifier [2]. Excessive condensation within the filter housing occurred on the ventilator side of the device. However, if the fluid is infected secretions on the patient side of the device, then the task of keeping the device dry may be more difficult.

The changes in airflow resistance, which we observed, were not predicted by whether breakthrough of fluid occurred. Water did not pass through the Hygrobac HME, but in soaking into the sponge material produced a dramatic increase in resistance. Water passed through the Edith and Intersurgical devices but only the Edith HME showed no marked increase in resistance. The important factor would seem to be whether moisture is actually retained in the media of the filter and is, therefore, presumably blocking the pores. We agree that a surface layer of water could prevent expiration but this is to some extent compensated in the Pall filter by its much larger surface area and pleated design.

It is true that the Pall device was designed primarily to be a filter. However, the hydrophobicity and physical properties of the filtering element also makes it an efficient condenser humidifier. There seem to be properties common to both and perhaps a company setting out to produce a condenser humidifier will inadvertently make an efficient filter!

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R.M. HEDLEY
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Faulty Touhy needle and dural puncture

We would like to describe a recent event that resulted in an inadvertent dural puncture and to which we believe a faulty Touhy needle contributed.

A middle aged woman was to undergo a hemihepatectomy for a solitary metastases following a gastric carcinoma. There were no contraindications to epidural block and this was performed prior to a general anaesthetic in the anaesthetic room. The midline approach at T₁₀ was chosen, but very quickly it was evident that a dural tap had occurred and the needle was removed. A further attempt was made to locate the epidural space at the space below, by a different operator, but with the same needle. Immediately it was noted that there was a leak from the needle (Fig. 1) and there was a loss of 'feel'. However, a successful epidural block was performed and the patient subsequently anaesthetised. She suffered no sequelae from

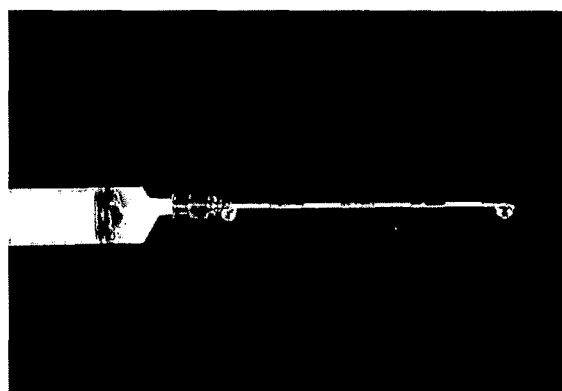


Fig. 1.

the dural puncture, although it was thought unsafe to use it for postoperative analgesia on the ward.

We believe that the leak from the E521 Everett 16 G Touhy needle contributed to the dural puncture by decreasing the change in loss of resistance with normal saline. The defect was not visible on inspection nor was it obvious unless injection through the needle was against resistance. We concluded that as with all anaesthetic equipment the Touhy needle should be tested for leaks prior to its use and that a visual inspection is not sufficient.

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A.T. WILSON
L. HAWTHORNE

A reply

Thank you for the opportunity to respond to the letter from Drs Wilson and Hawthorne. The authors describe an inadvertent incident where an E521 Everett 16 G Tuohy Needle may have contributed to dural puncture.

The problem appears to be that this particular needle

had a leak at the hub/needle blade junction which was not apparent to the initial operator as a loss of 'feel'. Some 2 years ago, our supplier improved the method of 'crimping' the hub to the needle blade in response to an earlier report of inadequate security, which was not associated with inadvertent dural tap [1].

Although batch identification was not possible in the case reported by Drs Wilson and Hawthorne, we believe that the needle predates this improvement. Certainly, there have been no similar reports concerning needles made with this enhancement. The product complies fully with the relevant British Standard, BS6196: 1990.

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Transdermal hyoscine and confusion in the elderly

We read with interest the conclusions of Dr P. Semple *et al.* (*Anaesthesia* 1992; 47: 399–401) in their paper regarding the use of transdermal hyoscine patch for antiemesis in patients using postoperative patient-controlled analgesia. We have had equally disappointing results using transdermal hyoscine in conjunction with postoperative fentanyl/bupivacaine epidural infusions. In addition, we noted the predictable but significant side effect of postoperative agitation and confusion, which drastically reduces the applicability of this method of antiemesis in the elderly.

We chose to study 30 patients (age range 54 to 80 years) presenting for either total hip or total knee replacement. They were randomly allocated into one of two groups of 15 each. Patients in group 1 had the hyoscine patch (Scopoderm TTS, CIBA) applied one hour pre-operatively, while those in group 2 received placebo. Premedication consisted of temazepam 10–20 mg administered orally 1–2 h pre-operatively. A standardised combined spinal/epidural technique was used and intra-operative sedation provided with propofol ($2 \text{ mg.kg}^{-1}.\text{h}^{-1}$). Postoperative analgesia was maintained by epidural infusion, with a mixture of 0.25% bupivacaine with fentanyl $25 \mu\text{g.ml}^{-1}$

($1\text{--}3 \text{ ml.h}^{-1}$). The fasting regimen was standardised and all patients were offered breakfast on the first postoperative morning. They were assessed for 48 h postoperatively for nausea and vomiting and also on a simple 10 point orientation scale (name, date, place, etc). Patients with persistent nausea and/or vomiting were given prochlorperazine 12.5 mg by deep intramuscular injection. In the hyoscine patch group, three patients experienced some degree of nausea and/or vomiting, compared to six in the control group. This was not statistically significant. Urinary retention occurred in four patients in each group, and these patients required catheterisation. Disorientation occurred in six out of 15 patients of the hyoscine group, while this was not seen in the controls. This was both clinically and statistically significant ($p < 0.01$).

This was not an entirely unexpected finding in this group, and despite the theoretical advantages of using this simple and noninvasive delivery system, we feel that transdermal hyoscine is both ineffective and unsuitable for use in this situation.

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M.R. CHECKETTS
D.M. COVENTRY

Propofol in hyperkalaemic periodic paralysis

A recent article (*Anaesthesia* 1992; 47: 579–84) described the safe use of thiopentone to induce anaesthesia in four patients who had the rarer of the two forms of potassium-associated familial paralysis. We report a patient with this condition in whom propofol was used uneventfully in this condition.

A 22-year-old female student was admitted for excision of a small fibroadenoma of the breast. She was known to suffer from familial hyperkalaemic periodic paralysis for which she took hydrochlorothiazide 25 mg daily. Pre-operatively her serum potassium was 3.1 mmol.l^{-1} . She was premedicated with diclofenac 100 mg and metoclopramide 10 mg orally. Following intravenous administration of fentanyl $75 \mu\text{g}$ and droperidol 0.75 mg, anaesthesia was induced with propofol 120 mg and maintained with a propofol infusion, supplemented by nitrous oxide in oxygen via a face mask. She received

230 mg of propofol in total. There were no intra-operative problems and immediately postoperatively her potassium was 4.1 mmol.l^{-1} . Recovery was uneventful and she was discharged from hospital the following day.

The use of propofol in familial periodic paralysis has been described only in the more common hypokalaemic form [1]. We believe this to be the first report of the safe administration of propofol in the more rare hyperkalaemic variant.

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Reference

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Transient ischaemic attack following carbon dioxide retention due to intrathecal morphine

A 76-year-old man with a history of chronic obstructive airways disease, angina and three previous transient ischaemic attack (TIAs) developed respiratory depression after pleurectomy. Pre-operative blood gases breathing air were as follows: pH 7.34, P_{aO_2} 10 kPa, P_{aCO_2} 4.3 kPa. He received an anaesthetic consisting of propofol, atracurium and nitrous oxide in oxygen supplemented by isoflurane. Analgesia was provided by intrathecal morphine 2 mg injected at the level of L_{3-4} after induction. Peri-operative management was uneventful and the patient went to the recovery ward following tracheal extubation.

He was pain free, fully awake and had a respiratory rate of 18 min^{-1} with an oxygen saturation of 99% breathing 40% oxygen. He was returned to the thoracic ward as all high dependency and intensive care beds had been taken up by emergencies. Written instructions to monitor respiratory rate and level of sedation half hourly were issued.

He was transferred to the high dependency unit 3 h later when a bed became available. He was found to be heavily sedated, responding only to pain and the right arm was paralysed. Respiratory rate was 20 min^{-1} , and breathing was shallow and uncoordinated. Oxygen saturation was 95% breathing 40% oxygen. He had pinpoint pupils, a good pulse volume and a blood pressure of 120/75 mmHg. A 12-lead ECG showed sinus rhythm with multiple ventricular ectopics. He responded to 400 μg naloxone intravenously with an increased respiratory effort and dilated pupils. Neurological examination confirmed loss of power in the right arm with loss of reflexes. Blood gases showed a respiratory acidosis (P_{aCO_2} 8.6 kPa and P_{aO_2} 16.4 kPa). His conscious level again deteriorated but responded to further naloxone. He was transferred to the intensive care unit where he stayed overnight breathing spontaneously, treated with naloxone and doxapram infusions. Overnight the P_{aCO_2} progressively decreased from 8.8 to 4.5 kPa. As his respiratory function improved

so did the function of his right arm, which by the following morning was completely normal.

This patient developed respiratory failure following what was in retrospect a rather large dose of spinal morphine [1]. Of particular interest is the nature of the transient ischaemic attack (TIA). The patient had a compromised cerebral circulation. Further enquiry showed that his previous TIAs gave rise to similar episodes of right monoparesis indicating a localised area of cerebral hypoperfusion. A possible explanation for the TIA is that cerebral vasodilation due to hypercapnia may have induced a condition of vascular steal as described by Brawley [2]. However, one recent study [3] found no evidence of steal during hypothermic cardiopulmonary bypass in patients with cerebrovascular disease. Other more mundane explanations, such as transient hypotension or cerebral emboli, may explain the neurological signs. The correlation of improvement in the neurological state and reduction in P_{aCO_2} suggests intracerebral steal as the underlying pathophysiology.

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Low-flow anaesthesia systems, charcoal and isoflurane kinetics

Since washout kinetics of inhalational anaesthetics are longer with low-flow anaesthesia systems, it is generally accepted that they are better suited for prolonged surgery where the 'margin of safety', in terms of time, is longer than short procedures. A proposed 'rule of thumb' is that for every hour of anaesthesia, anaesthetic vapours should be stopped 15 min before the anticipated end of surgery [1] (i.e. 45 min for a 3 h operation). However, this is too risky to apply in practice because of the risk of too early awakening. Fortunately, washout kinetics can be greatly accelerated by inclusion of a charcoal filter into the breathing system.

Twenty subjects (age: 51.6, SD 14.5 years), scheduled for extra and intracranial surgery (duration: 171.2, SD 63.5 min; minimum: 90 min-maximum: 340 min) were studied. All patients were premedicated with diazepam 0.15 mg.kg^{-1} orally or hydroxyzine 50 mg intramuscularly. After 5 min pre-oxygenation, anaesthesia was induced with fentanyl 3 $\mu\text{g.kg}^{-1}$, propofol 1.5 mg.kg^{-1} and atropine 0.01 mg.kg^{-1} . Tracheal intubation was facilitated by vecuronium 120 $\mu\text{g.kg}^{-1}$. Mechanical ventilation was regulated to maintain an end-tidal CO_2 4.0-4.5 kPa with a respiratory rate of 8-12 breaths.min^{-1} , a tidal volume of 10 ml.kg^{-1} and I:E ratio $\geq 1:2$. A mean total fresh gas flow (oxygen and nitrous oxide) of approximately 0.7 l.min^{-1} was used during surgery, with an end-tidal isoflurane concentration of 0.8-1.2%.

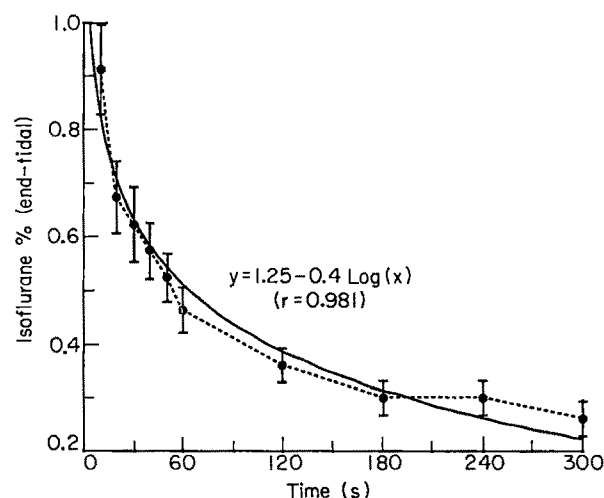


Fig. 1. End-tidal concentration of isoflurane (mean, SD), during the first 5 min after the introduction of charcoal cartridge into the low-flow circuit. The experimental washout curve is accurately described by a logarithmic curve: $y = 1.25 - 0.4 \log_{10}(x)$.

At the end of surgery, all anaesthetic gases were stopped and the inspiratory flow was bypassed through a small cartridge, prefilled with 40 g of charcoal (BDH Ltd, Poole,

UK), using the plastic sheet of a filter, Sterivent (DAR, Mirandola-Modena, Italy). Inspiratory and expiratory (end-tidal) isoflurane concentration was measured every 10 s with an Ohmeda RGM 4250 monitor, precalibrated with a gas mixture of known concentration (Anesthesia Calibration Gas, Scott Medical Products, Plumsteadville, PA, USA).

The inspiratory concentration of isoflurane declined to very low values (0.065 SD 0.049%) during the first 60 s, decreasing to zero in the following min; end-tidal concentration of isoflurane declined to MAC_{awake} values, according to Gaumann *et al.* [2], within 5 min of the introduction of the charcoal cartridge. The tracheas of all patients were extubated within this short time interval. The experimental washout curve of end-tidal isoflurane concentration can be accurately described by a logarithmic curve of the form: $y = a \cdot \log(x)$ (Fig. 1).

We used each charcoal cartridge for a maximum of four patients: in a separate, preliminary study, it was our impression that more prolonged use is accompanied by a progressive, rapid decline of the efficacy of charcoal. The

use of small-volume charcoal cartridges during low-flow anaesthesia, as suggested in this study, make low flow systems more flexible in their use: anaesthetic gas can be stopped at the very end of surgery, and the patients are completely awake in a few minutes. The anaesthesia level is also more easily controllable and accidental overdosing can be immediately controlled.

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Bacteraemia following nasopharyngeal airway insertion

Nasopharyngeal airways are frequently employed in medical practice. While it has long been established [1, 2] that nasotracheal intubation causes a transient bacteraemia, thus warranting appropriate antibiotic chemoprophylaxis in patients with cardiac lesions, no such data exist about the use of nasopharyngeal airways. We therefore read with interest the recent article by Ali *et al.* which reported that the use of oral and nasopharyngeal airways is not associated with bacteraemia (*Anaesthesia* 1992; 47: 153–5) and would like to report the results of a study which confirms their findings.

Thirty-five patients (ASA 1) who were undergoing minor surgical procedures under general anaesthesia using a mask were studied. They were assigned to have either a nasopharyngeal airway (study group) or an oropharyngeal airway (control group) inserted. Swabs from the anterior nares were taken from all patients. Ten ml of blood were sampled from an antecubital vein following skin preparation with 70% isopropyl alcohol 30 s and 3 min following insertion of the airway. Five ml were inoculated into both an aerobic and an anaerobic blood culture bottle ('Bactec system'). The bottles were screened twice daily for 48 h and thereafter once daily for 5 days. Isolates from nasal swabs grew predominantly normal flora, i.e.; *Staph. epidermidis*, *Staph. aureus* and *Strep. viridans*. No organisms were isolated from any of the blood cultures.

The aetiology of bacteraemia after nasotracheal intubation is thought to be related either to entry of nasopharyngeal commensals into the bloodstream via the

nasal mucosa during passage of the tube or to transfer of nasopharyngeal commensals from a contaminated tracheal tube into the bloodstream via the tracheal mucosa. The finding that insertion of a nasopharyngeal airway does not result in bacteraemia weakens the former theory; rather it lends weight to the theory that it is the presence of a contaminated tube in the trachea allowing passage of organisms through the tracheal mucosa which is responsible for bacteria entering the bloodstream.

In conclusion, we found that nasopharyngeal airway insertion, in contrast to nasotracheal intubation, is not associated with a bacteraemia. This coincides with the findings of Ali *et al.*; however, unlike these authors, we see no reason to recommend antibiotic prophylaxis for nasopharyngeal airway insertion in high risk patients.

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Anaesthesia for motor cortical mapping

The requirements of anaesthesia for intracranial surgery include a perfectly still patient who does not cough or strain on the tracheal tube, and a 'slack' brain. In addition the patient should wake up quickly after surgery so that a neurological assessment can be performed. These aims are usually achieved by using a technique of neuromuscular paralysis with light general anaesthesia. However, for motor cortical mapping, muscle paralysis cannot be used. Techniques that have been employed include awake craniotomy with sedation and local anaesthesia, or general anaesthesia without muscle paralysis. The latter requires

fairly deep anaesthesia to ensure no movement or coughing when the dura is open and the concentration of volatile agent required may produce a swollen brain unless hypocapnoea is induced by hyperventilation [1]. Recovery from anaesthesia will also be delayed. In order to achieve the desired conditions for surgery we used a technique of general anaesthesia with artificial ventilation of the lungs. For the initial part of the procedure a conventional relaxant technique was used, but during the period of cortical mapping, we used an alfentanil infusion to suppress breathing and coughing.

A 66-year-old man with a 9 month history of headaches followed by left sided arm and facial weakness, presented for craniotomy, motor cortical mapping and excision of tumour. He was otherwise healthy. Following premedication with glycopyrronium 0.2 mg 1 h pre-operatively, anaesthesia was induced with thiopentone 5 mg.kg⁻¹ followed by vecuronium 0.1 mg.kg⁻¹. Alfentanil 1.0 mg was administered prior to tracheal intubation and anaesthesia was maintained with isoflurane 0.5% and nitrous oxide with 35% oxygen. An alfentanil infusion was commenced at 0.5 mg.kg⁻¹.h⁻¹ and no further relaxant was required until the motor cortical mapping was completed. The expired CO₂ concentration was maintained at 3.5% using a Cape Wayne ventilator. Prior to motor cortical mapping we tested for residual neuromuscular blockade using a peripheral nerve stimulator, which demonstrated a train-of-four ratio of 1 and the absence of tetanic fade. Movement of the patient's hand during cortical stimulation confirmed recovery of neuromuscular transmission. When the cortical mapping was completed the patient was re-paralysed with vecuronium and the alfentanil infusion rate was halved initially and discontinued 30 min prior to reversal. Good operating conditions were achieved and the patient woke up quickly at the end of surgery and was alert during recovery.

Our use of alfentanil allowed ventilation of the lungs without neuromuscular blockade and without the risk of moving or coughing, while avoiding the problems of deep inhalational anaesthesia. Alfentanil does not increase cerebral blood flow [2] and because of its pharmacokinetic profile, recovery is rapid following discontinuation of the infusion [3]. These features make it ideal for intracranial surgery. This use of alfentanil has not been described before for this purpose and we feel it has much to recommend it in this type of case.

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Caudal anaesthesia in the Shy-Drager syndrome

We wish to report the successful use of caudal anaesthesia in a patient with the Shy-Drager syndrome which consists of progressive autonomic failure and multiple system atrophy [1]. A search of the literature has not revealed a previous report.

A 55-year-old man (height 1.79 m weight 84.5 kg) with Shy-Drager syndrome presented for cystoscopy. He gave a 6-year history of increasing difficulty in emptying the bladder with frequency and dribbling causing great distress. Other symptoms were loss of libido, impotence, progressive weakness of the left side of the body and Parkinsonism. Cystoscopy under topical anaesthesia had been abandoned due to inadequate analgesia. The patient had previously undergone several operations under general anaesthesia. These included exploratory laminectomy and extirpation of the ligamentum flavum at the L₃–L₄ level; implantation of a Scott penis prosthesis, and elongation of the tendon of the left big toe. Since the last operation 18 months earlier, his condition had deteriorated.

Relevant findings on examination were difficulty in swallowing with accumulation of saliva; shallow breathing with weakness of the respiratory muscles and coarse crepitations over both lung fields; inability to cough effectively and breathlessness on gentle exertion. Pulse rate was 70 beat.min⁻¹ and blood pressure 200/110 mmHg, but readings varied with posture. Caudal anaesthesia was considered appropriate.

Premedication was with diazepam 10 mg orally 1 h before surgery. Pulse and blood pressure were monitored throughout and an intravenous infusion of 2.5% dextrose with sodium 70 mmol.l⁻¹ (Ringerdex) commenced. A

caudal block was performed and 0.5% bupivacaine 20 ml and 2% mepivacaine 10 ml with adrenaline 5 µg.ml⁻¹ injected. The level of analgesia was confirmed as T₁₂–L₁ using an ethyl chloride spray.

During the first 20 min after injection of the local anaesthetics, systolic blood pressure decreased from 240 to 220 mmHg, and the pulse rate rose from 70 to 80 beat.min⁻¹. The patient was placed in the lithotomy position and this was followed by a rise in systolic pressure to 240 mmHg. As he found the position uncomfortable, diazepam 10 mg were given intravenously. Cystoscopy was then performed. On returning the legs to the horizontal, systolic pressure decreased to 210 mmHg although the pulse rate remained steady at 75–80 beat.min⁻¹. Postoperative recovery was uneventful.

In the Shy-Drager syndrome autonomic reflexes are defective resulting in an 'auto-sympathectomised' state. Regional blockade is thus likely to have only minimal effects on arterial blood pressure and this was illustrated by our patient.

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Nutricath insertion—a hazard

We wish to draw your attention to a hazard involving the insertion of a Vygon Nutricath 'S'.

A 63-year-old woman presented for replacement of a prosthetic aortic valve. A 16-G Nutricath 'S' 2180.17 was *in situ* in the right subclavian vein. It had been used for

4 weeks to administer antibiotics for treating an episode of subacute bacterial endocarditis. On removing the Nutricath, it was noted that 6 cm of stylet protruded from the end of the catheter. (Fig. 1) Review of the chest X-ray taken immediately after catheter insertion revealed that the

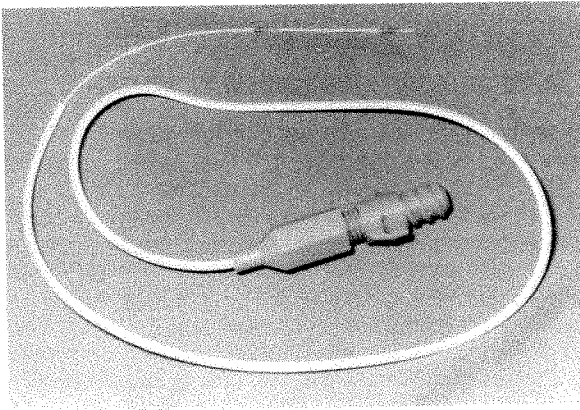


Fig. 1.

protruding stylet was visible. Comparison with a new Nutricath 'S' showed that the stylet was complete and that it had separated at the point of connection with the hub used for withdrawing the stylet. We were surprised to find that it was possible to inject fluid without undue difficulty down the catheter with the stylet in place.

The hub and stylet may not have been joined at manufacture. Alternatively the hub and stylet may have been weakly joined and when separated the operator failed to appreciate that the stylet remained in the catheter. The manufacturers' instructions specify that the stylet should be withdrawn an equivalent amount for every 5 cm of the catheter advanced. It is difficult to remove the stylet once the catheter is fully inserted and this may result in breakage.

Only four of 20 Vygon Nutricath 'S' catheters examined in this hospital came with enclosed instructions. Complications associated with leaving the stylet *in situ* include: embolus of the stylet; clot formation on the stylet,

bacterial growth and emboli. The rigid stylet might increase the risks of vessel wall erosion.

In conclusion, it is essential for the operator to familiarise him/herself with the manufacturers' instructions and the contents of the equipment about to be used. Careful review of the postinsertion chest X ray would have revealed the stylet.

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A reply

Thank you for the opportunity to comment on this unique incident.

We fully endorse the authors' conclusion that operators must familiarise themselves with the Instructions For Use (IFU) of any medical device. In addition we would recommend that anyone attempting the insertion of a tunnelled catheter of this type would do so initially under supervision of an experienced operator. Examination of the product reveals no evidence of a manufacturing defect. The history of the incident supports breakage of the stylet through user error.

The observation that comprehensive IFU are present in only four of the catheter sets is a result of updated IFU being introduced to this product range in mid 1990. Prior to this only limited instructions were included. Should anyone require additional copies we are able to supply them through Mrs Hazel Selby in our Customer Services Department on 0285 657051. An instructional video is also available on loan.

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Equipment deadspace in paediatric breathing systems

I am writing to remind anaesthetists of a potential hazard regarding the use of nonpaediatric equipment in children.

As receiving anaesthetist I took over the care of a child whose lungs were being ventilated through a T-piece and Jackson Rees modification. The system contained additional deadspace due to the inclusion of a disposable catheter mount and an adult sized condenser humidifier filter, inserted between the tracheal tube and the breathing system (Fig. 1).

The child was a 6-year-old boy who was transferred paralysed and his lungs ventilated because of a head injury, which had resulted in three grand-mal fits followed by an unrousable state. He weighed approximately 25 kg and therefore his estimated anatomical deadspace would be 52.5 ml ($7 \times 25 \times 30\%$). However, as his trachea was intubated this figure would be too high an estimate and could probably be halved. The measured equipment deadspace was 59 ml for the condenser humidifier and 27 ml for the catheter mount. Thus the total functional deadspace was 112.25 ml ($52.5/2 + 59 + 27$) or 213.8% of that expected in a child with an alveolar deadspace of 122.5 ml ($7 \times 25 \times 70\%$). The resultant blood gases at 6 l.min^{-1} of oxygen were pH 7.122; P_{CO_2} 11.45; kPa P_{O_2} 62.35; kPa HCO_3^- 28.0; mmol.l^{-1} ; B.E. -4.3 mmol.l^{-1} . Twenty minutes after the extraneous parts of the system had been removed, with ventilation being continued at 6 l.min^{-1} , they were: pH 7.452, P_{CO_2} 3.93 kPa, P_{O_2} 71.5 kPa, HCO_3^- 20.5 mmol.l^{-1} , B.E. -1.3 mmol.l^{-1} .

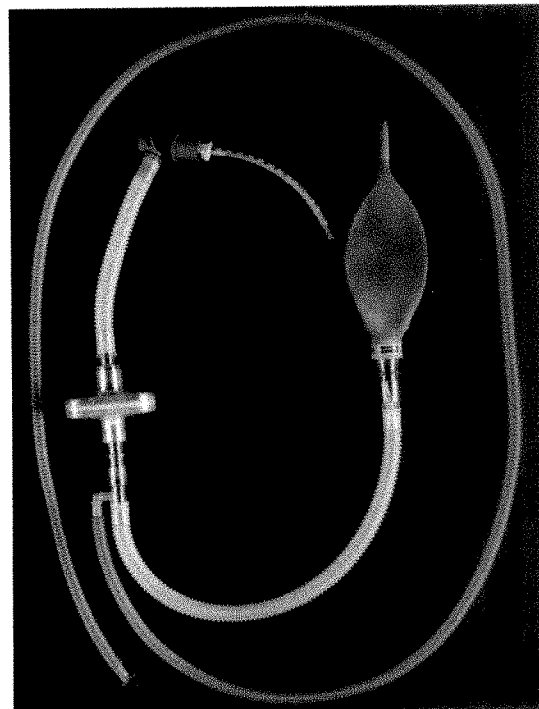


Fig. 1.

At follow-up the child was well. The fits were not due to severe head injury, and the unrousable state was apparently postictal. The potential consequences in a child with a severe head injury and developing cerebral oedema could have been disastrous. If a condenser humidifier must be

added it should be appropriate for the size of the child and without the catheter mount.

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L.M. Fox

The human wedge

I read with interest the article 'The human wedge', describing an important manoeuvre to relieve aortocaval compression during resuscitation in late pregnancy (*Anaesthesia* 1992; 47: 433-4). I found it unusual that the picture presented would depict the supine patient wedged with the right side downward for a number of reasons. Clinically, I have always moved the uterus to the left in order to avoid caval occlusion, while recognising that this may predispose to pressure on the aorta. This may reduce uterine blood flow (and fetal oxygenation) but would tend to augmentation of maternal blood pressure, cardiac filling and peripheral resistance, all desirable during maternal resuscitation. The aorta may be more resistant to compression than the inferior vena cava due to higher intravascular pressure (when present) and to the intrinsic fibromuscular structure of the vessel itself. During resuscitation, the return of maternal haemodynamics is paramount and I would argue that wedging to the right may still allow some caval compression.

The authors suggested that chest compression was 'better' under wedged conditions, because the incidence of correct compressions was 66.6% vs 32.5% when delivered strictly supine. The most common cause of incorrect

compression was reportedly due to excessive force. I would ask if it is possible that these differences in 'correctness' may be systematic errors in the study design. Perhaps the real message is that compression force is inherently less in the wedged position. This question needs to be answered and the proper techniques systematically taught. Basic life support training courses routinely teach techniques of infant, child and adult management. It is time to incorporate the specific technique of life support of the fourth 'type' of human, the term pregnant female, into all training programmes.

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The Editor replies

The author of the human wedge amended his paper at the proof stage to indicate that the left lateral was the preferred position. However, because of an oversight this information was not included in the final version.

Capnography and broncho-oesophageal fistula

We report a patient in whom the use of a capnograph enabled a diagnosis of broncho-oesophageal fistula to be made during the course of a rigid oesophagoscopy under general anaesthesia.

A 55-year-old woman with carcinoma of the oesophagus was scheduled to undergo rigid oesophagoscopy and oesophageal dilatation under general anaesthesia. After pre-oxygenation, anaesthesia was induced using a rapid sequence technique with cricoid pressure. Induction was with propofol and suxamethonium and maintenance with nitrous oxide, enflurane and vecuronium. Orotracheal intubation was performed using a 7.5 mm I.D. 'south facing' tracheal tube and capnography was commenced using a Datex Normocap capnograph (Datex Division Instrumentarium Corp., Helsinki, Finland).

At oesophagoscopy an encircling stricture was seen at the level of 18 cm from the lips. The surgeon (G.J.) commented that he could see bubbles formed by gas emerging via the lumen of the oesophagus from below the stricture. The origin of this gas was discussed. If this was coming from the stomach the intention was to go ahead with dilatation of the stricture, if from a broncho-oesophageal fistula, then oesophageal dilatation would be contra-indicated. The sampling tube from the capnograph was passed down the lumen of the rigid oesophagoscope and the capnograph showed the presence of CO₂ in the gas emerging from the lumen of the stricture. This, however,

was not diagnostic, as it is well known that gastric gas contains CO₂. To further investigate this situation, the inspired gases were altered to contain 8% CO₂ for a few breaths. Within two breaths, there was a marked change in the readings displayed by the capnograph; instead of cycling between 0% and 5% it was now cycling between 8% and approximately 6%, and these changes were reversed when the CO₂ was stopped. It was thus possible to say with confidence that there was a broncho-oesophageal stricture and the investigation was terminated at this point, with no dilatation being performed. The rest of the anaesthetic, and recovery were unremarkable.

We believe that this is the first time that the pre-operative diagnosis of a broncho-oesophageal fistula with a capnograph has been reported. The advantage of added safety with capnography during general anaesthesia can be augmented by using it to trace the path of respiratory gases in patients with abnormal anatomy. It is worthy of note that this is only possible using an anaesthetic machine with which the use of CO₂ is still permitted. In the absence of this, one might consider using a volatile agent meter to perform a similar function, although in this case there was not one available.

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Capnography and awareness

We wish to report two cases in which problems were caused during general anaesthesia by inadvertent depression of the emergency oxygen flush button. Both cases involved emergency Caesarean section and in both instances a pre-

anaesthetic check of the Boyles International machine found everything to be in order, including the oxygen flush button which correctly delivered high flow oxygen.

In the first case anaesthesia was induced with a rapid

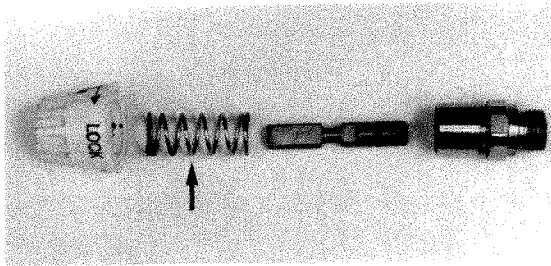


Fig. 1.

sequence induction using thiopentone 5 mg.kg^{-1} and suxamethonium 1 mg.kg^{-1} . The trachea was intubated and the lungs ventilated with 1% isoflurane in 70% nitrous oxide and oxygen using a Nuffield Penlon 200 ventilator and a Bain coaxial system. Fentanyl $100 \mu\text{g}$ was given after delivery of a healthy male infant. Fifteen minutes later the end-tidal carbon dioxide was noted to be 2.9 kPa . As all other vital signs including pulse and blood pressure were entirely stable, hyperventilation was diagnosed and ventilation was adjusted accordingly. However, the end-tidal carbon dioxide continued to decrease and a further cause was sought. Disconnecting the Bain system from the machine revealed a faint hiss from the swivel connector. The oxygen flush button was found to be partially depressed and the problem was rectified. The inspired isoflurane concentration was increased and intravenous hyoscine was given in an attempt to increase sedation and amnesia. The patient was not sweating or lacrimating unduly. Chest movements and inflation pressures were normal and there was no tachycardia or change in blood pressure. At the end of the operation the oxygen flush button was examined and it was found that the return spring (arrowed, Fig. 1) was missing from the assembly. The machine had recently returned from a service and we presume that the return spring had been omitted during re-assembly. The patient experienced some awareness of events after induction but did not feel any pain.

The second case involved a similar method of anaesthesia. However, soon after tracheal intubation and connection to the Bain system and Nuffield Penlon ventilator, the combination of high airway pressures and low end-tidal carbon dioxide (1.6 kPa) was noted. At the same time chest movements were seen to be large. Institution of manual ventilation confirmed a high fresh gas flow and inspection of the Boyles machine rapidly revealed that in this case the oxygen flush button was locked in the 'on' position; no awareness occurred.

Cases of awareness during general anaesthesia as a result of the emergency oxygen flush button remaining locked in the 'on' position are well reported [1–3]. A typical scenario is that at some stage the button is leant against and an

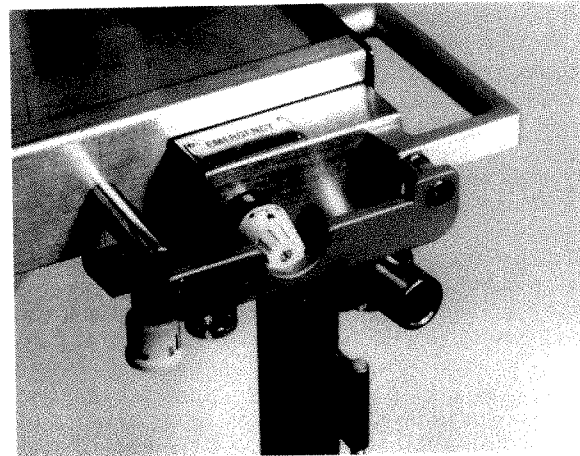


Fig. 2.

additional rotational movement allows it to become locked, as probably happened in the second case described. Using a ventilated Bain system, the high fresh gas flow usually produces exaggerated chest movements and abnormally high inflation pressures which help to identify the problem, as in case two.

In our first case the fault was much more subtle, and in the absence of an inspired oxygen analyser, only capnography gave a clue to the problem. To our knowledge only one author [3] has previously drawn attention to the usefulness of capnography in these circumstances. Cooper cites the overlapping absorption bands for nitrous oxide and carbon dioxide as the cause for an elevation of end-tidal carbon dioxide readings obtained during inhalational anaesthesia [3]. Once the nitrous oxide has been largely displaced with high flow oxygen, it is suggested that the resultant readings will be relatively low, hence indicating a problem. We believe, in our case at least, that the explanation is more simple and results from the oxygen flush button causing continuous hyperventilation. In an effort to prevent this type of incident occurring again we have fitted a protection bar in front of the oxygen flush button (Fig. 2).

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No need to modify the Sprotte needle

Agilan and Stansby (*Anaesthesia* 1992; **47**: 506–7) propose a modification to the Sprotte needle. The excellent clinical characteristics would not be affected if the lateral eye of the Sprotte needle were to be reduced to the size of the internal diameter. The authors maintain that this modification results in greater stability to the tip of the needle as well as a reduced risk for inadequate spinal anaesthesia.

However, my modification to the Whitacre needle was carried out quite deliberately, with the lateral eye to considerably exceed the physically requisite size for laminar flow. There were two reasons behind this. Firstly, even with

correct positioning in the subarachnoid space, the cauda equina and arachnoid may well obstruct, either completely or partly, the eye of the needle. 'Dry' taps and uncertainty as to whether the needle has entered the subarachnoid space have their own risks and tend to increase the failure rate of spinal anaesthesia. Together with the extra thin wall, the apparently large lateral eye of the Sprotte needle makes for rapid flow of cerebrospinal fluid (CSF), even if the eye is partly obstructed. This characteristic of the needle enables diagnostic lumbar puncture to be performed more frequently at the first attempt, with an incidence of

postlumbar puncture headache comparable to that of spinal anaesthesia [1, 2]. Secondly, the elongated lateral eye of the Sprotte needle is more suitable for preventing inadvertent intraneural injection in neural blockade procedures. These definite advantages of the large lateral eye have to be taken into account when considering the theoretical drawbacks referred to by Aglan and Stansby.

With regard to the alleged greater failure rate, this was not apparent during spinal anaesthesia in the 6-year testing phase preceding initial publication [3]. A prospective random double-blind study revealed a significantly lower number of incomplete blocks for the 24-gauge Sprotte needle compared to a 25-gauge Quincke needle (1.6% vs 7.8%) [4]. Furthermore, routine use of the 24-gauge Sprotte cannula in a number of neuroradiological departments over a period of several years indicated that there were no problems with double epidural-spinal injection of contrast media during myelography, i.e. with free CSF aspiration, the entire contents of the syringe are distributed solely in the subarachnoid space. Naturally, the stability of the needle tip is greater if the lateral eye is smaller. Therefore a special quality steel is employed for the production of these needles to compensate for the lateral eye and reduced wall thickness [4].

For use in the USA the FDA require comprehensive physical testing. To avoid any misunderstandings it should be pointed out at this juncture that for comparative purposes we were forced to refer to 22 and 25-gauge Whitacre needles from Becton and Dickinson, which

despite their smaller lateral eye proved to be more unstable than the 22 and 24-gauge Sprotte cannula (Test Certificate of TÜV Product Service Abt. Chemie, Medicalprodukte Munich 18.06.91).

I should like to express my thanks to Drs Aglan and Stansby for their constructive contribution, which discusses one critical aspect of the Sprotte needle and at the same time offers me the opportunity to comment on this publicly.

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Intravenous magnesium therapy in critically ill patients

Magnesium homeostasis [1-3], the use of magnesium in the control of tachyarrhythmias in critically ill patients [4, 5], and the effect of magnesium on mortality following acute myocardial infarction (LIMIT-2 study) [6], have been the subject of several recent reports. A number of different dosing schedules have been documented. Some regimens suggest fairly rapid infusion of magnesium sulphate (20 mmol (5 G) over 1 min [4], 8 mmol (2 G) over 5 min [6]), while others are more circumspect: 40 mmol (10 G) over 24 h [3].

Using a 'rapid' regimen (8 mmol.5 min⁻¹), we administered magnesium sulphate to three patients suffering from Gram-negative septicaemia, who were subject to a variety of tachyarrhythmias (atrial fibrillation, nodal/supraventricular tachycardia, recurrent ventricular ectopy). In two patients, hypotension (due to a fall in systemic vascular resistance (SVR) and requiring increased inotropic support) persisted for over 4 h. A third patient again showed a fall in SVR but unaccompanied by hypotension because of a significant increase in cardiac output. The hypotensive response was not unexpected although we were surprised by its duration, particularly as hypotension had not been a significant problem in the LIMIT-2 study [6] whose dosage schedule we were using. Despite this, in all three patients, the arrhythmias were significantly blunted.

Since these experiences, we have altered our regimen, so that magnesium (48 mmol) is infused over 24 h.

Hypotension has not been noticeable. We suggest that in septicemic patients (who are subject to global myocardial depression, unlike most patients suffering from acute myocardial infarction), magnesium should be infused slowly, unless there is clear evidence of clinical hypomagnesaemia (e.g. fasciculation, hypotonia etc).

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Ten minutes wasted—you're lucky!

Having read the letters titled 'And where does the time go during operating lists?' (*Anaesthesia* 1992; 47: 69-70 and 630), I felt I must contribute from the system in which I currently work.

Like the above authors, I find considerable variation in

time taken to induce anaesthesia; the similarity ends here though. We have no anaesthetic rooms, therefore patients have to be brought into the operating theatre after it has been fully prepared, which causes considerable delay. Once in the operating theatre monitors are applied and the

search for the surgeon begins. Some are very good about arriving with the patient, but as there are no strict operating lists, surgeons find themselves doing clinics, etc. and will not leave their other commitment until the patient is in the operating theatre, thereby minimising the time they waste. During induction there is a tradition that the surgeon attends apparently 'to get the anaesthetist out of trouble.' I think this comes from the not too distant past of nonmedical anaesthetists. The surgeon now supervises the positioning of the patient and indicates to the scrub nurse which part of the anatomy to clean. He/she now leaves the operating theatre to scrub and returns, hands held high, to be gowned and gloved by the scrub nurse.

For many months I have anaesthetised routine cases and have never timed tracheal intubation to skin incision at less than 30 min, the average being nearer 40 min. It certainly does not require a mathematician to see that our days are not only long, but that we do half the number of cases of similar UK hospitals. This means more hospitals *per capita*. Now one can see where the health budget goes.

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Anaesthetists, nurses and pre-operative visiting

I share Drs Severn and Morris' concern (*Anaesthesia* 1992; 47: 645) about the place of pre-operative visits by operating theatre nurses without prior consultation with the anaesthetic team. Wires can all too easily become crossed and ill-considered advice proffered producing a confused and bewildered patient. We have recently had practical experience of such a situation when the play therapists at our children's hospital decided to 'play out' anaesthetic experience with young children who were to undergo surgery, without first seeking the advice of the anaesthetists. Children were shown a 'teddy' having an anaesthetic mask pushed onto its face and syringes with 18 gauge needles were used to demonstrate injecting 'teddy'.

As a result, we were presented with terrified children arriving in the anaesthetic room some of who were impossible to reassure.

Whilst I would not wish to discourage the operating theatre nurses' interest in any aspects of patient treatment, I doubt that it is of benefit to the patient. However, if pre-operative visits by nurses do take place it must be with a member of the anaesthetist team responsible for that patient.

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The Humphrey ADE, Magill and Lack breathing systems

It has been suggested (*Anaesthesia* 1990; 45: 341) that carbon dioxide elimination using intermittent positive pressure ventilation (IPPV) with the Humphrey ADE system in the Mapleson A mode would be inefficient; a modification to overcome this 'problem' was proposed. The A semi-closed anaesthetic breathing systems are, traditionally, unsuitable for IPPV because fresh gas may be lost through the expiratory, or pop-off, valves; these can be closed with each inflation of the lungs, but the technique is usually impractical. Hand ventilation with the ADE system in the A mode has been evaluated previously in adults, although the system used was described as similarly modified [1], and has been independently evaluated in children [2]. However, the Mapleson A systems are not identical. We expected that there might be differences in the rate of CO₂ elimination between various A semi-closed systems employed in this way.

The Humphrey ADE in the A mode, Magill and Lack systems were tested in patients undergoing general anaesthesia requiring neuromuscular blockade to determine whether end-tidal carbon dioxide ($P_{E'}\text{CO}_2$) increased with hand ventilation. The tracing shown (Fig. 1)

was obtained during surgery for above-knee amputation in an 81-year-old man. Initially, IPPV was provided with a Manley ventilator set at tidal volume 600 ml and fresh gas flow of 5.5 l.min⁻¹. This gave a reading of 12 breath.min⁻¹ on a Datex Cardiocap which sampled at 150 ml.min⁻¹ from a port between catheter mount and tracheal tube. When a steady state was established, the ventilator was replaced by hand-controlled IPPV using each system in turn. The anaesthetist was blind to the tracing being made and the patient was supervised by an assistant. Fresh gas flow was unchanged, the displayed respiratory rate was kept at 11–13 breath.min⁻¹ and the expiratory valves were adjusted to achieve a constant chest expansion. Between each breathing system the ventilator was replaced for 2 min and recording suspended.

We found that end-tidal carbon dioxide increased during IPPV using the Magill attachment. Unexpectedly, $P_{E'}\text{CO}_2$ did not rise with either the ADE or the Lack system, but instead tended to decrease towards or below the values with mechanical ventilation. A possible explanation for this is that the deadspace at the patient end of these two systems is relatively small and end-alveolar gas is cleared

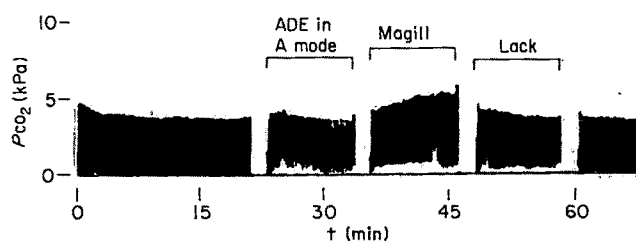


Fig. 1. Carbon dioxide partial pressures in IPPV with three breathing systems.

easily by fresh gas. No modification of the Humphrey ADE system was necessary as IPPV in the A mode appeared to be effective.

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Tracheal tube cuff pressure measurement—inexpensive continuous monitoring

Ischaemic injury of the tracheal epithelium in intubated patients is directly proportional to tracheal tube cuff pressure. Tracheal damage is unavoidable when the pressure exerted by the cuff exceeds mucosal perfusion pressure (20–35 mmHg) [1], while insufficient cuff inflation exposes the patient to aspiration of oropharyngeal or gastric contents. Nitrous oxide, by its physical properties, diffuses into the cuff and can cause substantial variations in cuff pressure. Most anaesthetists estimate cuff pressure by finger palpation, but this is very subjective and often misjudges the inflation pressure [2]. Consequently, objective cuff pressure measurement is recommended, but objective pressure measurement devices are rarely part of anaesthetist's standard equipment.

In our institution, most operating theatres are equipped with Siemens Elema 900 (Siemens—Elema, Sweden) ventilators with two ventilating systems, manual and automatic. During IPPV, the manual circuit is disconnected and its manometer, graduated from 0–100 cmH₂O, can be connected directly to the tracheal tube cuff by a manometer tube (Fig. 1). By adding a three-way stopcock, cuff pressure can be adjusted with a

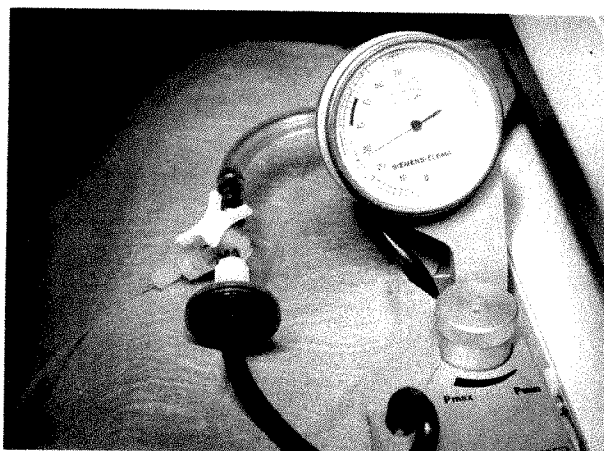


Fig. 1. The manometer of the breathing circuit with the manometer tube connected.

A leaking tracheal tube connector

I would like to report another cause of gas leakage from an anaesthetic breathing system. A 19-year-old woman had a general anaesthetic for instrumental vaginal delivery of twins. Her trachea had been intubated with a size 8 mm Curity tracheal tube (The Kendall Company (UK) Ltd, Basingstoke, Hampshire RG24 0WG) and her lungs were ventilated with a Dräger Narkomed 2B via a circle breathing system. I noticed a whistling noise emanating from the patient end of the system during inspiration. On

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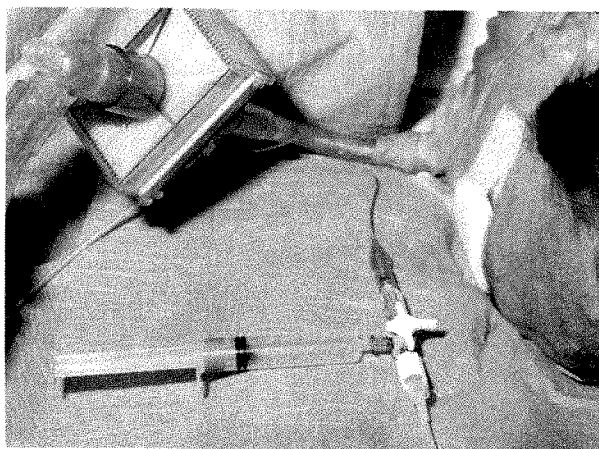


Fig. 2. The patient end of the set-up.

disposable 5 ml syringe (Fig. 2). When no direct contact with the patient's head is possible (neurosurgery, long lasting stomatology or ENT surgery), tracheal tube cuff pressure can be adjusted by placing this three-way stopcock proximal to the manometer.

This simple, reliable and inexpensive device can give continuous, objective information upon the pressure of the cuffs of either single or double-lumen tracheal tubes.

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close inspection of the tube and connector, I found a small hole in the connector. (Fig. 1) This hole was not covered by the tube and was allowing the escape of gas. At the end of the case, the plastic connector was found to be particularly brittle and shattered when manually compressed.

No other holes were discovered in the remainder of this particular batch of eight tubes, but a significant number of the connectors were brittle and splintered on manual compression. These particular tracheal tubes have been the

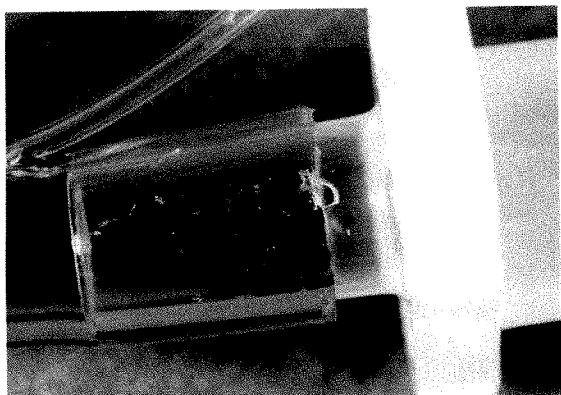


Fig. 1.

subject of a previous report [1]. However, on that occasion, the fault was at the proximal end of the tube rather than the connector. Although occasional faults will occur in mass produced disposable equipment [2], I was particularly concerned that this appears to have affected a whole batch of tracheal tubes.

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The Humphrey ADE breathing system and paediatric anaesthesia

We noted with interest the suggestion by Dr Humphrey (*Anaesthesia* 1992; **47**: 640–1) concerning the use of the Humphrey ADE breathing system to reduce pollution in the operating room during dental anaesthesia. We recently evaluated [1] the use of this system in children and would agree with the above proposal in that scavenging is easy with this system. Although we concluded that this system is suitable for use in children, we did not recommend its use, as claimed. We still feel that the Jackson-Rees modification of the Ayre's T-piece is the 'gold standard' for use in paediatric anaesthesia. We were also interested that Dr Humphrey found that hand ventilation in the A mode in adults worked particularly well (*Anaesthesia* 1992; **47**: 625). We found that in children hand ventilation in the A mode was less predictably efficient [1]. While the mean end-tidal partial pressure of carbon dioxide was 5.3 kPa, the range

was 4.1 to 7.7 kPa. Bearing in mind that switching to hand ventilation often occurs at more critical moments, higher fresh gas flows should be used in this mode during paediatric anaesthesia.

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Salbutamol via the laryngeal mask airway for relief of bronchospasm

We would like to report a method for administration of salbutamol via the laryngeal mask airway (LMA) for relief of intra-operative bronchospasm. A 63-year-old man scheduled for cystoscopy and with a history of chronic obstructive airways disease underwent general anaesthesia. Following premedication with temazepam, anaesthesia was induced with propofol and fentanyl and a size 4 LMA was introduced easily using the standard technique. Anaesthesia was maintained with nitrous oxide, oxygen and isoflurane, ventilation being spontaneous. Towards the end of the procedure it was noted that the patient had a markedly prolonged expiratory phase with straining. Auscultation revealed widespread wheeze despite an end-tidal isoflurane concentration of 1.2%.

A salbutamol aerosol (Ventolin inhaler: Allen and Hanbury) was connected to an 8 inch disposable Xylocaine spray nozzle (Astra B0643), the LMA was disconnected from the breathing system and the apparatus inserted down the LMA. Three metered doses (300 µg) were administered synchronously with inspiration and rapid improvement of the patient's condition resulted. Within 3 min there was no audible wheeze and the patient awoke breathing comfortably.

Before insertion down the LMA, a slight curve may be

moulded at the end off the spray nozzle, the length of which is sufficient to protrude beyond the mask aperture bars. Brimacombe [1] reported up to 70% of patients with the LMA *in situ* had some part of the epiglottis visible from the end of the LMA. Therefore it is probably best to keep the end of the nozzle 1–2 cm proximal to the mask aperture bars to allow maximal amount of bronchodilator aerosol to enter the distal bronchial tree. This manoeuvre would also minimize the theoretical risk of precipitating laryngospasm following deposition of aerosol on the vocal cords. If using a closed circuit breathing system or during intermittent positive pressure ventilation via the LMA, a connector with bronchoscopy cap may be used to achieve drug delivery.

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The role of the laryngeal mask in prehospital trauma care

I read with interest the two case reports on the use of the laryngeal mask airway (LMA) in prehospital trauma care by Greene, Roden and Hinchley (*Anaesthesia* 1992; **47**: 688–9). I am pleased that the LMA is increasingly being considered for emergency airway management when intubation of the trachea proves impossible. The degree of protection against aspiration provided by the LMA remains unproven, but as the authors stated, there is some evidence that it is, at least, partially protective [1].

In the prehospital care of trauma victims (and indeed other critically ill patients), adequate tissue oxygenation is the therapeutic priority. Therefore, in situations where tracheal intubation is not possible, the LMA is a useful alternative. It has been shown that paramedical personnel can easily learn to use the LMA and insert it rapidly with only a small failure rate (94% success rate at first insertion) [2]. The second-line intervention for airway maintenance in North America is the oesophageal obturator airway (OOA) and, to date, there have been no studies directly comparing the OOA with the LMA. However the OOA is associated with recognised complications (tracheal obturation, oesophageal perforation and aspiration of stomach contents on removal) and is not available in paediatric sizes [3]. The OOA also requires neck flexion for insertion and is contraindicated in cervical spine trauma, whereas the LMA can be inserted with no neck movement.

There has been one small field study examining the use of

the LMA in prehospital trauma care, but it only investigated 25 patients and there was no clinical information about these patients. However it did confirm the high success rate of insertion of the LMA in the field [4]. Insertion of the LMA is a very valid second-line manoeuvre when intubation of the trachea is not possible during prehospital emergency care. I believe LMAs should be available wherever advanced life support is carried out (including in ambulances) and that when paramedical personnel are taught tracheal intubation they should also be taught how to use the LMA.

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Laryngeal mask airway and low flow anaesthesia

We would like to describe our experience combining the Brain laryngeal mask airway and low flow anaesthesia. Twenty patients suitable for anaesthesia with a laryngeal mask airway were chosen. Following induction with propofol a laryngeal mask was inserted and its correct position ascertained. The patient was allowed to breathe spontaneously through a circle system with a carbon dioxide absorber. Anaesthesia was maintained with nitrous oxide, oxygen and isoflurane. A fresh gas flow of 4:2 l.min⁻¹ N₂O:O₂ was reduced after 15 min to 2:1 l.min⁻¹ N₂O:O₂, and then 5 min later to 1:1 l.min⁻¹ N₂O:O₂. This has been shown to safeguard against hypoxia developing in the circle due to the differential nitrous oxide and oxygen uptake by the patient. No case lasted more than 2 h. End-tidal carbon dioxide concentration was measured with a calibrated Datex capnograph and in none of the patients increased to more than 6%. Oxygen saturation was measured with a Novamatrix 505 and did not decrease below 95%.

This technique combines the advantages of a laryngeal mask airway with those of a circle system. The economical aspect of this arrangement should be emphasised as the cost of the volatile agents used in the main operating theatres of a district general hospital (£56 000 per annum) could be halved by the use of a low flow technique in the operating theatre and normal high flows in the anaesthetic room [1].

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Nil by mouth after midnight?

We modified pre-operative fasting guidelines at Foothills Hospital, Calgary, Alberta, Canada in 1988 following publication of the studies quoted by Dunnett and Zorab (*Anaesthesia* 1992, **47**: 638). The Department of Anaesthesia agreed unanimously that instructions for ambulatory (day surgery) patients should be modified to read:- 'Do not eat any solid food after midnight. On the morning of surgery you should drink a cup (6 oz = 150 ml) of coffee or tea, or a glass (6 oz = 150 ml) of apple juice or water 3 h before your scheduled time of surgery.' In a follow-up study of 211 patients [1], 73% had taken

advantage of the relaxed guidelines. The remaining 27% were not thirsty, thought the drink might cause nausea or vomiting postoperatively, or their surgeon had insisted on the old guidelines.

Change of guidelines for inpatients was more difficult because of nurses following the last order to be written. If a surgeon wrote the order 'NPO after midnight' later than the anaesthetist wrote 'clear fluids until 3 h pre-operatively' the surgeon's order was followed and *vice versa*. To overcome this confusion, the heads of the Departments of Anaesthesia and Surgery wrote a joint letter to all members

of their Departments and to all head nurses (sisters) which recommended no solids after midnight and unrestricted clear fluids until 3 h before the scheduled time of surgery. In a follow-up study of 199 inpatients [2], 105 ingested an average of 240 ml. The remaining 94 patients did not drink, either because they were sleeping from midnight until 3 h before the time of their surgery, or because they did not want to drink; only 16 patients were prevented from drinking by their surgeons or nurse. These two follow-up studies [1, 2] confirmed that there was no difference in gastric fluid volume or pH between those patients who were allowed to drink and those who fasted from midnight.

The new guidelines have been well received by patients and nursing staff. The fear by some anaesthetists that, if the absolute fasting rule were relaxed, patients could not be trusted to drink only clear fluids but would feel free to eat and drink anything they wished, has not been realised. Patients do occasionally admit to having eaten a full meal. The frequency is no greater than before the change in guidelines and the patient's usual explanation is that no instructions were given; whether there are clerical errors or the patients have not read the instructions in these instances is uncertain. Such occurrences are rare and are a minor problem for anaesthetists compared with the frequent discomfort of patients due to unnecessary deprivation of oral fluid [3, 4].

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References

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We were interested in the letter from Drs Dunnet and Zorab discussing the 'nil by mouth after midnight' rule (*Anaesthesia* 1992; **47**: 638). They question the justification for prolonged pre-operative fluid deprivation imposed on elective surgery patients and their Department is considering changing policy in this area.

After reviewing the literature in 1990, anaesthetists, paediatricians and nurses in both the University Hospital and the City Hospital, Nottingham, agreed on a policy of free clear fluids for children up to 3 h pre-operatively. The child is offered a clear fluid drink 3 h before surgery. Clear fluids include water, carbonated drinks, clear fruit juice, tea or coffee (with a small amount of semi-skimmed milk and sugar permitted). An anaesthetist is consulted regarding patients who are taking drugs known to delay gastric emptying and the rules do not apply to children with a disease known to delay gastric emptying or increase acid production. Patients are not given any food from midnight

if on a morning operating list, but can have a breakfast at 0700 if scheduled for surgery in the afternoon. Water 0.5 ml.kg^{-1} may be taken with oral drugs up to 1 h pre-operatively.

Having instituted this policy, there have been no complications to our knowledge and we believe children are less fractious, being less thirsty on arrival in the anaesthetic room. We are probably still being over cautious. Schreiner *et al.* [1] have shown no increase in gastric volume when children are given free fluids up to 2 h pre-operatively compared with a group who received no fluid for 6 h (or 8 h if over 5 years), even though most received oral premedication of pethidine, diazepam and atropine.

This year, following a 6 month pilot scheme on one ward, the traditional guidelines have been changed for adults as well. They are now last solid food 6 h pre-operatively and last drink 3 h pre-operatively; similar rules apply to types of fluid. Again, this may be over-restrictive, but has the merit of fitting in with nursing schedules and giving drinks at 0600 h makes it easier for routine medication to be given on the day of operation. Patients on afternoon operating lists are given a light breakfast at 0800 h and a morning drink at 1100 h. In practice, because of difficulties in gauging when patients are to go to the operating theatre, the 3 h rule is often applied to the time of the start of the operating list. This still avoids the very prolonged fluid deprivation previously enforced and there is, in general, strong support from the nurses. Again, no complications have so far resulted from this change in policy.

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Reference

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Rather more than 20 years ago I stopped giving intramuscular premedication on the day of operation other than to patients who were in pain. Since then I have insisted that all patients are allowed to drink and receive food until at least midnight and that all patients between 0600 and 0700 h receive a cup of tea or coffee and a modest quantity of toast for operating lists commencing after 0830 h. For those on an afternoon operating list the same procedure is followed except that they are given a drink about 1100 h. I have never had cause to regret this practice. To the best of my knowledge I have not had an aspiration and ward staff tell me that the patients are more comfortable in the immediate postoperative phase and tend to have less nausea and vomiting than would be expected. They also tend to eat at least some of the next scheduled meal unless this is contraindicated for surgical reasons.

The only difficulty that I have had is in ensuring that this regimen is carried out, as nurses are still being taught in the old-fashioned way. It is, I fear, necessary to explain gently, but firmly, at least once a month in the majority of the wards concerned that patients who are not given their breakfast will not be operated on, with as straight a face as is humanly possible.

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Like Drs Dunnet and Zorab (*Anaesthesia* 1992; 47: 638), we too became interested in the origins of the 4 to 6 h fluid restriction that has become a standard requirement before elective anaesthesia. A search of the literature was unfruitful and standard anaesthetic texts, while all extolling the need for this nil by mouth period, did not provide any references.

We undertook a prospective, randomised controlled trial in 100 elective surgical patients. We allowed free clear oral fluids until 2 h prior to induction in the study group. The gastric volume and pH was no different than that of patients who had been nil by mouth. Also noteworthy was the fact that the mean fasting duration in the control group was 14.9 h. Even those keen to maintain the current practice must agree this is excessive. Patients who had been allowed to drink were markedly less thirsty [1]. This trial only allowed benzodiazepine premedication.

The solution of this problem is more difficult than simply reducing the fasting time required. At present the evidence suggesting a reduction in fasting interval is not broad enough for a universal policy change. The studies reported to date have been retrospectively controlled and cautious with regard to the volumes of fluid allowed [2-4]. All but one have avoided opioid premedication [5]. This study, while finding that drinking had no effects on gastric contents, only allowed 150 ml of water. The use of drinks containing milk and sugar has also been untested. Introducing a policy with too many 'ifs' is sure to fail.

Nevertheless, the Canadian Anaesthetists' Society revise their guidelines for fasting in 1990, reducing the recommended fluid free interval before surgery to 3 h [6]. They emphasise the fact that a stomach can never be guaranteed to be empty and practice should consider the age, medical condition and type of surgery.

We feel that allowing elective surgical patients to drink until 2 h prior to induction of anaesthesia enhances patient comfort, reduces operating theatre delays and cancellations and does not compromise safety. However, the weight of evidence is not yet great enough to allow a blanket change of policy.

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Errata

Anaesthesia, 1992, Volume 47, page 819–820

Atracurium block prolonged by low dose tubocurarine

M. R. NOTT AND B. J. POLLARD

In the third line of the third paragraph of this letter the sentence should read:

Recovery of tone occurred first in only 10 patients, between 30 min in two pyrexial subjects and 2 h 5 min.

Three sentences later it should read:

Additional injections of atracurium 5 or 10 mg provided 20 to 30 min further relaxation.

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Corporate author

The Committee on Enzymes of the Scandinavian Society for Clinical Chemistry and Clinical Physiology. Recommended method for the determination of gamma-glutamyltransferase in blood. *Scandinavian Journal of Clinical Laboratory Investigation* 1976; **36**: 119–25.

Anonymous. Epidemiology for primary health care. *International Journal of Epidemiology* 1976; **5**: 224–5.

BOOKS AND OTHER MONOGRAPHS

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OSLER AG. Complement: mechanisms and functions. New York: Prentice-Hall, 1976.

Corporate authors

American Medical Association Department of Drugs. *AMA drug evaluations*, 3rd edn. New York: Publishing Sciences Group, 1977.

Editor, compiler, chairman as author

RHODES AJ, VAN ROOVEN CE, comps. *Textbook of virology: for students and practitioners of medicine and other health sciences*, 5th edn. Baltimore: The Williams & Wilkins Co., 1968.

Chapter in book

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Agency publication

National Center for Health Statistics. Acute conditions: incidence and associated disability, United States, July 1968–June 1969. Rockville, MD: National Center for Health Statistics, 1972. (*Vital and health statistics, Series 10: Data from the National Health Survey*. No. 69) [DHEW publication No. (HSM) 72–1036].

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